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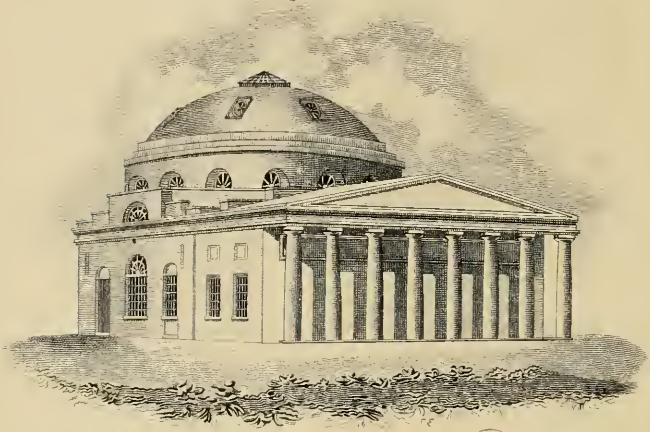

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THE CLINICAL AND PATHOLOGIC MANIFESTATIONS OF INFECTIOUS HEPATITIS*†

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Infectious hepatitis has been designated as one of the most important diseases of World War II, ranking with malaria and venereal diseases as a cause of morbidity. This disease, which has existed for many years, has a world wide distribution and has been a scourge in many previous wars. The thousands of cases of infectious hepatitis which occurred in epidemic form in the military forces focused medical attention on this entity (4). The stimulus thus engendered has resulted in partial clarification of the concepts concerning the sporadic cases and the small epidemics of hepatitis occurring in the civilian population.

This disease has previously been known as "epidemic catarrhal jaundice" and "non-spirochetal infectious jaundice". It has been suggested that the term "catarrhal jaundice" as applied to the sporadic case, be replaced by "infectious hepatitis" and that the term "infectious epidemic hepatitis" be used to designate an outbreak of a group of related cases. The close association of the conditions previously known as catarrhal jaundice, epidemic jaundice and acute yellow atrophy was noted and discussed many years ago and has been re-emphasized once again in the light of recent epidemiologic and pathologic disclosures (22, 8).

Substantial evidence has been accumulated to warrant the assumption that this disease is caused by a specific filterable virus (13). This virus will

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withstand a temperature of 56 C. for one-half hour, storage in a dry state at 0 C. for many months, and will remain viable in a liquid state for almost as long. It resists chemical agents in the usual concentrations in which they are used to purify water. This virus agent has not yet been grown in the laboratory, and experimental animals have not been successfully infected. The lack of a suitable laboratory animal to which the disease could be transferred has necessitated the use of human volunteers to study the resultant artificially acquired hepatitis (23).

Transmission of this disease to humans can be accomplished by the oral ingestion or by the subcutaneous or intravenous injection of filtered extracts of blood, feces, urine or duodenal juice from a spontaneous case or from another artifically infected human volunteer. This is particularly true if the material to be administered is collected from the known case during the preicteric stage or shortly after the appearance of jaundice. The incubation period is usually three to five weeks.

The enteric-oral route is the most tenable one explaining the natural means of transmission (21). Several reports describe the finding of the infective agent in the well water of a camp and associate this with the fact that the water had been contaminated by the excreta from camp patients. It has also been suggested that contaminated food, such as milk, may have been the vehicle of transmission in occasional epidemics. Unrelated individuals, who have eaten at the same restaurant during a certain period, have been known to acquire the disease after about one month. Droplet infection has been suggested as a means of natural spread of this disease (12). Volunteers have been infected by spraying the nasopharynx with material obtained from the nasopharyngeal washings of infected individuals. Family outbreaks of this disease are common; clinical symptoms appear first in one member and then about three to five weeks later, in other members.

Homologous serum hepatitis is a closely related entity which warrants consideration in a discussion of infectious hepatitis (24). This condition follows the injection of blood or its derivatives contaminated with an icterogenic virus agent, either closely related to or identical with the agent causing sporadic and epidemic infectious hepatitis. This condition has occurred following the administration of whole blood, fresh, dried or frozen plasma or serum. Pooled plasma, with a single donor's plasma containing the specific icterogenic agent, can produce the disease. This has necessitated the additional inquiry of prospective blood and plasma donors as to whether or not they have had jaundice.

Homologous serum hepatitis was first brought to the attention of the medical world by the cases of jaundice occurring three to six months after individuals had been inoculated with a certain yellow fever vaccine containing human serum, contaminated with an icterogenic virus. This condition has been shown to be entirely unrelated to yellow fever, and since human serum is no longer employed in preparing yellow fever vaccine, this particular source of homologous serum hepatitis has been eliminated. Also, this symptom complex has occurred after the injection of measles and mumps convalescent serum and in diabetic and venereal disease clinics where syringes, contaminated with the serum of preceding patients have been used without heat sterilization. The injection of as little as 0.01 cc. to 0.005 cc. of contaminated serum has been reported as sufficient to cause this disease. Various reports suggest that 2 per cent to 5 per cent of the casualties during the past war who received pooled plasma, later developed homologous serum hepatitis after three to six months. In the civilian population it has been estimated that less than 2 per cent of the individuals receiving pooled plasma develop the condition.

There has been much discussion concerning the relationship of infectious hepatitis and homologous serum jaundice since the clinical manifestations are strikingly similar. Whether they are variants of the same disease or separate but similar disease processes remains to be determined. This will be clarified only by a more definite analysis of the etiologic virus agent or agents, which are either identical or very closely related. Thus far, the physical characteristics of the virus of infectious hepatitis and homologous serum hepatitis are identical.

Certain apparent differences exist between these two conditions and are as follows:

(1) Cross immunization has not been found to occur. Patients have developed naturally acquired infectious hepatitis after having had homologous serum hepatitis resulting from the administration of yellow fever vaccine a year prior. Also, volunteers who had recovered from homologous serum hepatitis and were then injected with material containing the virus of infectious hepatitis, developed this disease (20).

(2) The modes of transmission are quite different as has already been stated. Infectious hepatitis probably occurs naturally via the enteric-oral route and to a lesser extent, via the upper respiratory droplet route. Homologous serum hepatitis occurs following the injection of serum, plasma, or blood contaminated with the icterogenic agent. The virus of homologous serum hepatitis has not been transferred from the feces of cases to susceptible volunteers whereas this has been done repeatedly in infectious hepatitis.

(3) The epidemiologic spread of these two conditions is different. An outbreak of infectious hepatitis may be associated with secondary and tertiary cases throughout a community but this does not occur in homologous serum hepatitis since direct and natural spread from man to man has not been reported.

(4) The incubation period is three to five weeks in infectious hepatitis and is three to six months in homologous serum hepatitis.

(5) Although these two conditions are strikingly similar insofar as the clinical manifestations are concerned, homologous serum hepatitis is thought to be the more severe and to have a higher mortality rate than infectious hepatitis. The latter disease has a mortality rate of 0.2 per cent to 0.4 per cent.

The clinical manifestations of infectious hepatitis have been conveniently divided into three stages (25); The pre-icteric stage, the icteric stage and the final stage.

The pre-icteric stage usually begins insidiously after an incubation period of three to five weeks. It lasts one to fifteen days, usually about five to six days, may be mild or severe and is usually ushered in with gastrointestinal symptoms. Anorexia, particularly for fatty foods, nausea and vomiting may be present. Generalized abdominal discomfort and even abdominal pain and tenderness in the right lower and right upper quadrant may occur. Appendectomies have been performed on several of these patients in the pre-icteric stage, but usually the finding of an enlarged and tender liver will prevent this mistake. The spleen may be palpable and cervical, or even generalized, adenopathy may occur.

Chilliness, fever, headache, weakness and generalized malaise may be the only symptoms at this time but are usually associated with the aforementioned gastrointestinal symptoms. In some epidemics or even in the sporadic case a grippal respiratory onset may occur (12).

In the last third of the pre-icteric stage the urine becomes dark and is found to contain bilirubin. This finding can be used as a helpful guide to the correct diagnosis and to foretell the imminent onset of icterus. There is also an increase in urinary excretion of urobilinogen at this time because of the hepatocellular inadequacy to receive the urobilin returned from the gastrointestinal tract. This rise in urinary urobilinogen is the first phase of the diphasic curve of urobilinogen excretion. The second rise occurs with the recession of the icteric phase (28). The cephalin-cholesterol flocculation, the thymol turbidity, and the bromsulfalein tests may indicate liver damage in over 50 per cent of cases in the pre-icteric stage. The sedimentation rate is usually normal at this time and the total white blood cell count is not remarkable, but shows a tendency toward a reversal of the lymphocytic-neutrophilic ratio.

The icteric stage occurs in about 90 per cent of all cases of infectious hepatitis. When this disease occurs without the appearance of jaundice, and in this event it is clinically less severe, it is difficult to make the correct diagnosis unless it is suspected because of the presence of associated cases with jaundice. This stage usually lasts three to five weeks, and is roughly proportionate to the intensity of jaundice which may vary greatly. When jaundice occurs, the gastrointestinal symptoms are accentuated and the generalized non-specific constitutional symptoms may subside. Right sided abdominal pain and tenderness increase and the liver becomes larger and more tender. Enlargement of the spleen and lymphadenopathy may become more pronounced. The stools become light in color, the urine becomes darkly discolored by bilirubin and itching of the skin may be troublesome. At this time the Van den Bergh test may give a direct positive reaction and the mistaken diagnosis of cholelithiasis and cholecystitis is not infrequently made.

The icterus index may vary from 10 to over 200; values of 100 to 150 are not uncommon. Usually the icterus index begins to fall during the second to third week and is normal by the sixth or seventh week. A second increase in urinary urobilinogen excretion occurs with the recession of icterus. In the icteric stage the cephalin cholesterol flocculation and thymol turbidity tests are positive in a high percentage of cases, and the prothrombin time may be prolonged.

The final stage occurs in 0.2 per cent to 0.4 per cent of cases and these patients die with the symptoms and signs of so-called acute or subacute yellow atrophy (17). This stage frequently occurs in individuals who are debilitated and malnourished or alcoholic. Acute liver failure supervenes, the icterus becomes intense, the temperature rises, and the patient lapses into coma after a period alternating between excitement and drowsiness. The presense of ascites and the finding of an increased prothrombin time which does not respond to vitamin K therapy suggest an unfavorable prognosis.

The convalescent stage usually requires three to five weeks and sometimes longer following the return of the icterus index to a normal level. It is characterized clinically by a subsidence of symptoms and a regression in the size of the liver. Relapses will frequently occur if the period of convalescence, with full bed-rest and adherence to the dietary regime, is not continued long enough to effect a cure. A complete cure is characterized by a normal urinary urobilinogen output, a regain in weight and appetite, and an adequate fulfillment or cautiously graduated exercise tolerance tests. If cure has not been attained, the exercise tolerance tests will provoke a return of gastrointestinal symptoms, liver enlargement, an increase in urinary urobilinogen excretion, and, on occasion, jaundice.

There is a small percentage of individuals who despite the best of care may become what is known as chronic relapsers (14). They continue to show remissions and exacerbations of fatigue, vague gastrointestinal symptoms, an itolerance to fat and right upper quadrant discomfort. It is still quite controversial as to whether or not this chronicity can ultimately cause a diffuse hepatic cirrhosis.

The differential diagnosis of infectious hepatitis in the temperate zone includes a consideration of such conditions as grippe, atypical penumonia, infectious mononucleosis, enteric infections, leptospirosis, acute appendicitis, acute cholecystitis, and cholelithiasis.

The treatment of infectious hepatitis warrants the following considerations: (1) Early instituted and sufficiently prolonged bed-rest, (2) a high protein, high carbohydrate, high vitamin, and moderate to low fat diet, (3) avoidance of additional liver damage by abstaining from alcohol, and the prevention of exposure to secondary infections.

The administration of immune serum globulin during the incubation period serves as an effective temporary prophylactic agent against infectious hepatitis (11). This prophylactic effect does not hold true for homologous serum hepatitis (10).

There is marked variation in the severity of the clinical manifestations and also in the pathologic findings. However, that pathologic lesions differ quantitatively rather than qualitatively and are, therefore, basically the same, (18, 27, 9). The following 3 cases are presented to illustrate these variations in clinical course and pathologic anatomy.

The following case illustrates a fulminating form of hepatitis that is often called acute yellow atrophy. The hepatocellular destruction is complete and there is no regeneration of parenchymatous cells. The absence of regeneration is caused by the rapidity and the completeness of the necrotic process (17). Lucke and Mallory (18) described a number of cases occurring in the Army that were similar to this one. Most of the cases of that series were war casualties who received large amounts of plasma.

Case No. I (Chart #84960): A 50 year old colored housewife was admitted to the University of Maryland Hospital on August 18, 1946, in a semistuporous, delirious, condition. Her illness began acutely on August 14, 1946, with dizziness, vomiting, and headache. On August 17, 1946, she became very irritable and later irrational. On admission to the accident room she was found to be jaundiced.

Her past history revealed that she had typhoid fever in 1926, and a thyroid operation in 1945, at which time a toxid adenoma was removed. After that the patient enjoyed good health until the present illness. There was no history of alcoholism, syphilis, or of recent drug or serum therapy.

On admission her pulse rate was 100 per minute, temperature 98 F., and blood pressure 128 systolic over 75 diastolic. Stupor and malnutrition were noted. The skin was dry and inelastic, and marked generalized icterus was present. The epigastrium, particularly the right hypochondrium, was tender. However, the liver and spleen were not palpable.

Laboratory data: On August 18, 1946, her hemoglobin was 90 per cent, and the white blood cell count was 9,550 per cmm. The differential leucocyte count was normal. The blood sugar was 34 mg. per 100 cc. of blood, and the blood urea nitrogen was 9 mg. per 100 cc. of blood. The presence of urobilinogen in the urine was doubtful. On August 20, the blood sugar rose to 190 mg. after the administration of parenteral glucose. The quantitative Van den Bergh test indicated 3.6 mg. of bilirubin per 100 cc. of blood initially

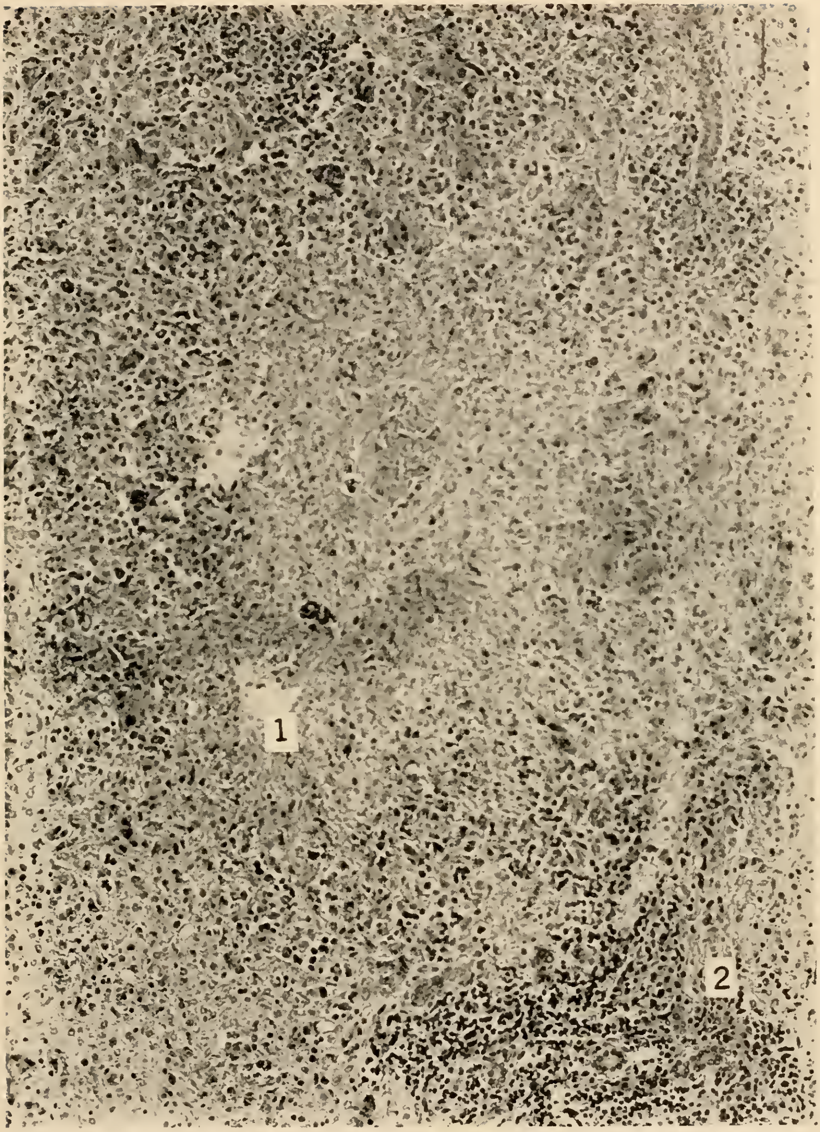

Fig. 1.—Case number 1. Complete hepatocellular necrosis.
1. Central veins.
2. Portal triads with cellular infiltration.

and 8.1 mg. after extraction with alcohol. The blood cholesterol was 291 mg. per 100 cc. and the cephalin flocculation test was 4 plus. A serologic test for syphilis and the urine and throat cultures were negative.

The patient was given small doses of paraldehyde in order to control the delirium.

She received parenteral glucose, multiple vitamins, and oxygen. Her temperature remained normal, but she became comatose and died forty hours after admission.

Autopsy findings: The abdomen was found to be free of fluid. The liver was small, weighing 750 grams. It was soft in consistency, and homogeneously pale in color. The lobular markings were indistinct. The gall bladder was filled with a small amount of dark green bile. The biliary system was patent throughout.

A microscopic examination of the liver revealed almost complete destruction of the hepatic cells (see Fig. 1). However, the lobular pattern was preserved, and there were a few scattered hepatic cells with pale cytoplasm and dark nuclei remaining at the periphery of some of the lobules. There was infiltration by monocytes, plasma cells, and lymphocytes with a few polymorphonuclear neutrophils. The cellular infiltration was most marked at the periphery of the lobules. There was some evidence of bile duct proliferation but no regeneration of the parenchymal cells was evident.

Case No. II (Chart #80650). A 21 year old white male was admitted to the University of Maryland Hospital on March 21, 1945, because of severe abdominal pain and vomiting. The patient stated that he had been jaundiced for about five weeks. On March 19 he developed generalized abdominal pain, nausea, and vomiting, but continued to work until the day of admission. There was no history of lues, ulcer pain, hematemesis, or occupational hazard.

On admission his temperature was 101.5 F., pulse rate 110 per minute, and respirations 45 per minute. The patient looked sick, was dehydrated and moderately jaundiced. He was in acute distress but well oriented. The heart was of normal size with regular rhythm and no murmurs. The blood pressure was 80 systolic over 50 diastolic. There was spasm of the abdominal muscles on the right side. The liver and spleen were not palpable. The percussion note was dull over the lower abdomen and flanks, with tympany in the epigastrium. His urine showed a positive test for bilirubin and the presence of 5 white blood cells per high power field on microscopic examination. The blood carbon dioxide combining power was 21 volumes per 100 cc., and the blood bilirubin 8.06 mg. per 100 cc. of blood, direct reaction, and 7.06 mg. indirect.

Under gas-oxygen-ether anesthesia the abdomen was opened revealing the presence of bile-tinged ascitic fluid. The gall bladder was distended. The tissues of the upper abdomen appeared "markedly edematous." The liver was dark red, mottled, and smaller than normal. The appendix was normal. A cholecystostomy was done. The patient died six hours after admission.

Autopsy findings:—The body was diffusely icteric. All other significant findings were confined to the abdomen. There was a recent right upper quadrant incision 10 centimeters in length and an adjacent stab wound from which several rubber drains protruded. The spleen weighed 1000 grams and measured 20 x 14 x 8 centimeters. There were areas of yellowish discoloration 1 to 8 millimeters in diameter seen through the capsule and larger similar areas were seen on the cut surface. The organ had a firm consistency but the yellow areas were doughy. The liver (see Fig. 2) weighed 1350 grams and measured 30 x 18 x 8 centimeters. The external surface was covered with slightly elevated yellowish nodes. The cut surface of the right lobe revealed areas of greenish-yellow discoloration. The rest of the liver was dark red in color. The terminal ileum, the caecum, and the ascending colon showed thickening, redness, and edema.

Microscopic examination of the liver revealed large areas of complete hepatocellular destruction. In other areas there was less destruction of liver cells. Many of the cells were swollen and granular, while others were large with pale cytoplasm and prominent nuclei, (see Fig. 3). These cells represent parenchymatous regeneration (17). There was a diffuse infiltration by lymphocytes, monocytes and plasma cells that was most marked about the lobular periphery and the portal triads. There was no evidence of fibrous tissue proliferation. The spleen displayed a distortion of the architecture with congestion, hemorrhages, and cellular infiltration by monocytes, lymphocytes, and plasma cells. There were many scattered small foci of necrosis. The intestinal wall was edematous with necrosis of the submucosa and areas of the muscularis. There was dense widespread polymorphonuclear infiltration (see Fig. 4).

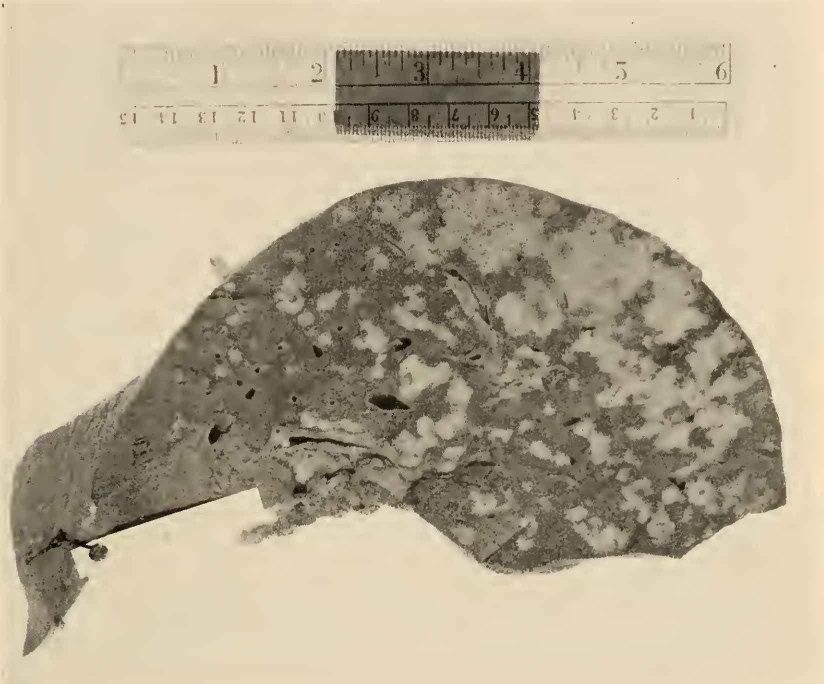

Fig. 2.—Case number 2. Hepatocellular necrosis with patchy regeneration.
1. Light areas are hepatic cells.
2. Dark areas are necrotic.

Case No. II illustrates a subacute variety of hepatitis. The clinical course and the liver damage is not as severe as in case No. I. There is proliferation of the remaining parenchymatous liver cells in those lobules where destruction was not complete. There is no evidence of cellular regeneration in those lobules showing complete parenchymatous necrosis. This explains the irregular areas of regeneration.

Investigators report an incidence of ascites varying from 25 to 60 per cent in fatal cases (18, 20). Ascites is thought to be related to both protein alterations and to congestive changes in the liver, (1, 27, 26).

Lucké (27) found a phlegmonous colitis in 15 per cent of a series of subacute hepatitis but none in the fulminating cases.

The clinical aspects of mild hepatitis, so called catarrhal jaundice, are familiar to every physician. The lesions in the liver are similar but less extensive, and not as marked as in the preceding cases. Necrosis, proliferation, and inflammatory reaction are the essential features. Necrosis begins

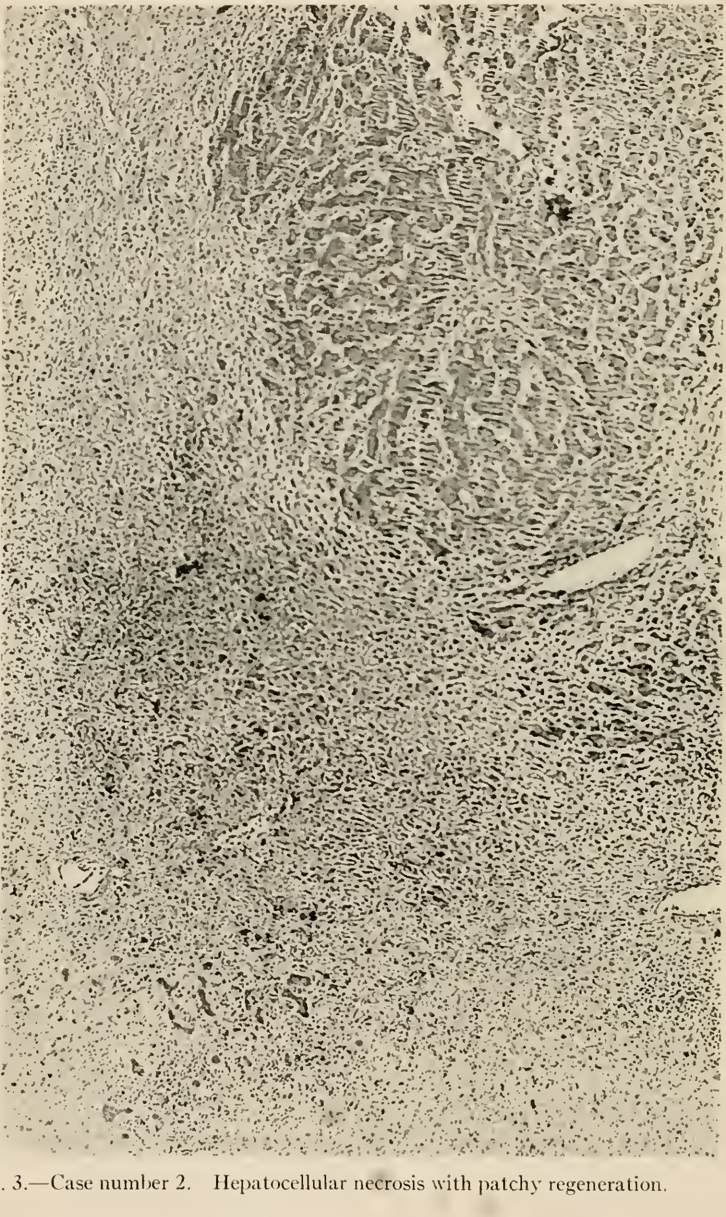

Fig. 3.—Case number 2. Hepatocellular necrosis with patchy regeneration.

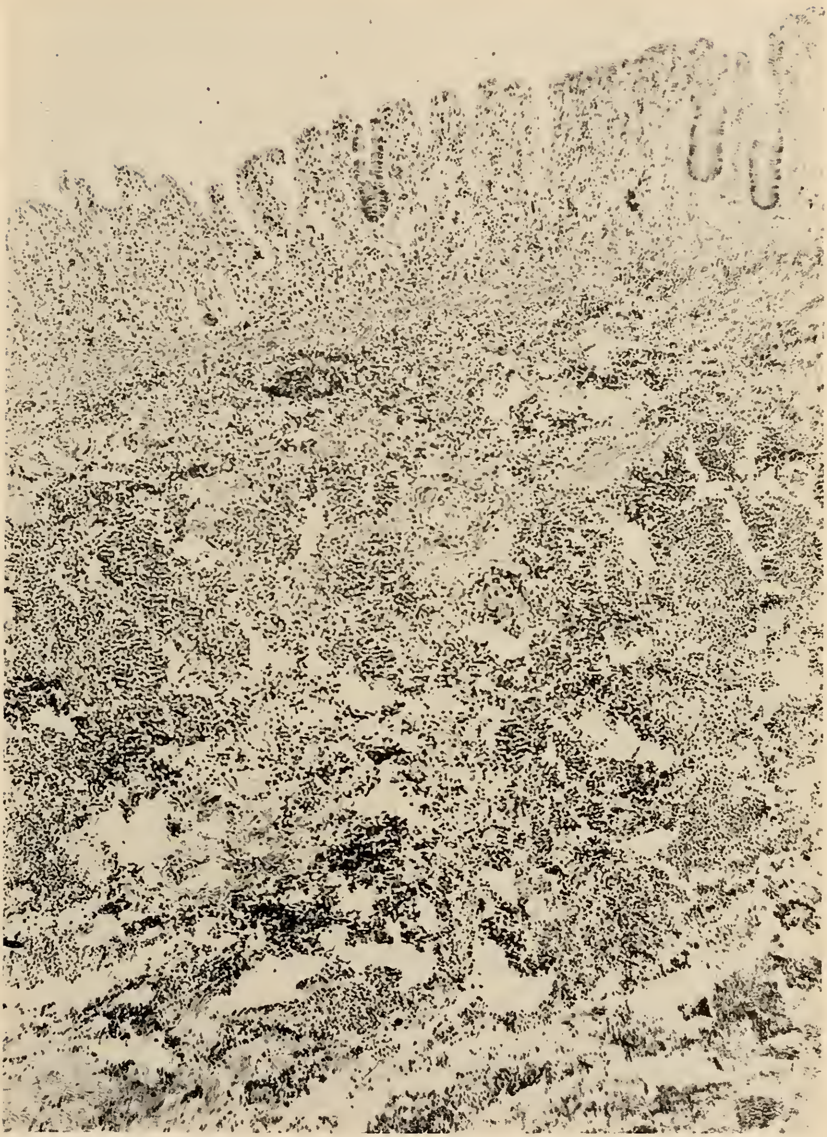

Fig. 4.—Case number 2. Phlegmonous colitis. This picture shows necrosis and dense polymorphonuclear infiltration of the entire wall of the colon.

and is most marked about the central vein (17, 18, 27, 9, 26), (see Fig. 5). Complete regeneration may take place from the hepatic cells remaining at the periphery of the lobule, (9, 26).

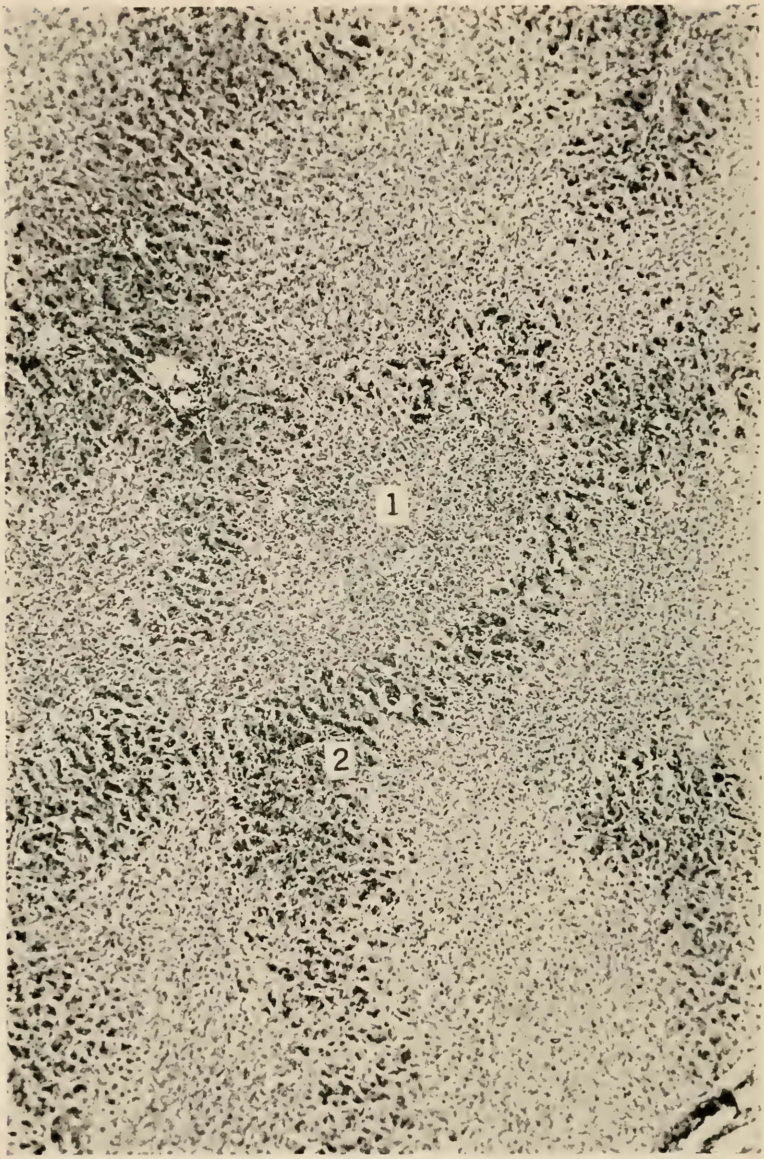

Fig. 5.—Necrosis most marked about central veins.
1. Areas of hepatocellular necrosis.
2. Parenchymal cells at the periphery of the lobules.

The prevalence of hepatitis in the Armed Forces has focused attention on the possible sequelae of this condition. Numerous investigators have found a diminution in the excretory functions of the liver after apparent recovery, (5, 3, 2, 6, 14). Case No. III illustrates the type of residual liver damage that may follow in a very small per cent of these cases. There is a complete loss of hepatic cells in many lobules with collapse of the reticular framework.

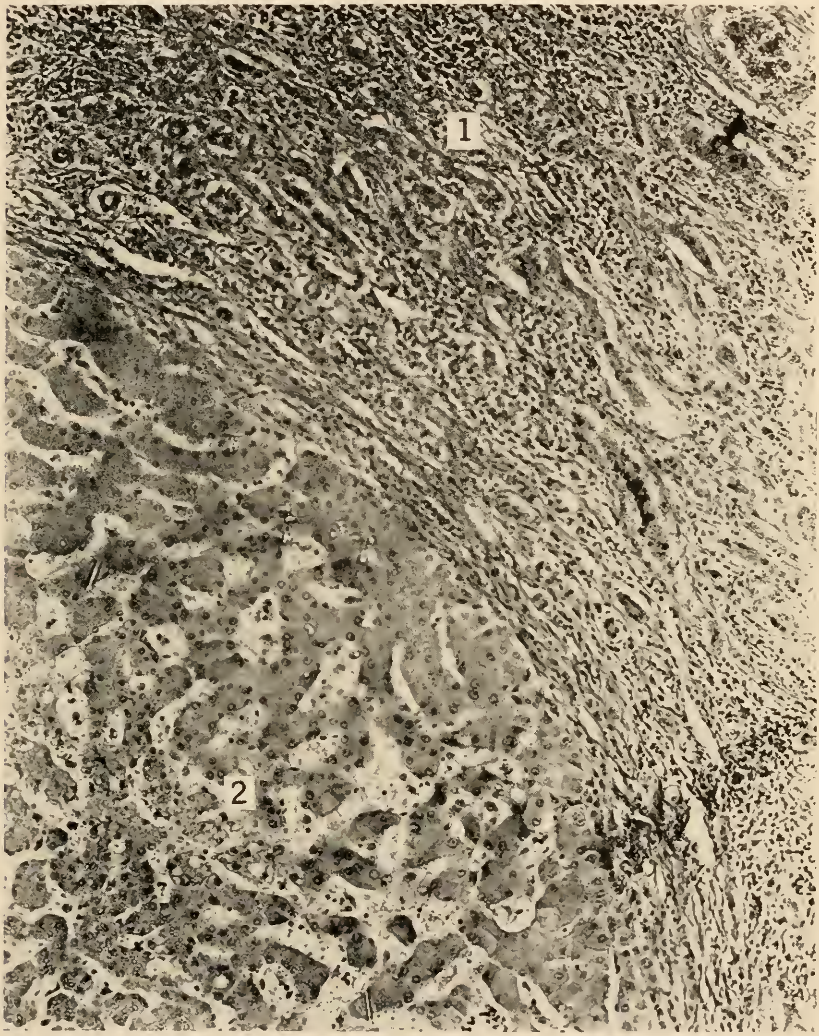

Fig. 6.—Case number 3. Late residual distortion of hepatic architecture.

1. Hepatic cells.
2. Collapsed and compressed reticular framework with proliferation and cellular infiltration.

Other lobules partially or completely regenerate from the remaining liver cells. The only real distortion is caused by the growth of the regenerating cells (see Fig. 6). There is very little, if any, increase in fibrous tissue (28, 17) but chiefly a condensation or collapse of the reticular fibers with rough preservation of the lobular liver pattern. Lichtman (15) and Lucké (16) believe this does not constitute a true cirrhosis. It is certainly not a portal cirrhosis (15, 19, 7) although it may give rise to a syndrome of portal hypertension and hepatic insufficiency.

Case No. III (chart #46647): A 21 year old white female was admitted to the University of Maryland Hospital on September 12, 1943, because of chills of one week's duration and a dry rasping cough that lasted two days. Two months before admission the patient noted swelling of her ankles and jaundice associated with an intolerance for fatty and greasy foods. Her past history revealed nothing of significance until a prior admission to the University Hospital in 1941 for painless jaundice. She had been jaundiced for six months prior to the admission in 1941, but she had no associated symptoms. Her clinical findings were those of mild hepatitis, and she was given a special diet and directions to be followed at home. The patient left school and restricted her physical activities but the jaundice persisted for a total of one year. She gained weight and was well until two months prior to the second admission to the hospital.

A physical examination on admission revealed a temperature of 102.8 F., a pulse rate of 110 per minute, and blood pressure of 130 systolic over 80 diastolic. She was well nourished and well developed. There was a marked generalized icterus. An examination of her chest revealed signs of pneumonia with pleural effusion on the right side which was confirmed by roentgenography. The heart was normal. The abdomen was rounded and tense and there was a fluid wave with shifting dullness in her flanks. There was tenderness along the right costal margin, but the liver and spleen were not palpable. There was a moderate amount of pitting edema of her ankles and feet. Her urine contained bilirubin but was otherwise negative. Blood counts were normal. Cultures of the blood and pleural fluid were negative. A culture from the throat showed Diplococcus pneumoniae and Staphylococcus aureus (hemolyticus). The serologic test for syphilis was negative. The blood non-protein nitrogen was 27 mg. per 100 cc., and her blood sugar was 53 mg. per 100 cc. The albumin was 3.28 grams and globulin 5.29 grams per 100 cc. of blood. The Van den Bergh reaction was prompt direct 13.6 mg. per 100 cc. The prothrombin time was 42 seconds with a control of 16 seconds. On September 20 a positive blood culture for Escherica coli was obtained.

The patient was given sulfathiazol, parenteral fluids, and multiple vitamins. Her temperature subsided but otherwise her condition remained unchanged. The temperature rose and she died on the eighth hospital day.

The autopsy was limited to inspection of her abdominal and thoracic viscera with removal of blocks of tissue for microscopic study. There was 300 cc. of bloody fluid in the right pleural cavity but inspection and microscopic examination revealed no evidence of pneumonia. The liver measured 19 x 11 x 8 cm., and was estimated to weigh 800 grams. It was pale yellow in color and studded with numerous firm, raised, nodules varying in size from 1 to 4 mm. The cut surface of the liver seemed to reduplicate the external surface. The biliary system was patent, and no stones were found.

A microscopic examination showed oval nodules of remaining liver parenchyma. These were surrounded by collapsed reticular framework and proliferating bile ducts. There was a patchy infiltration of monocytes, lymphocytes, and plasma cells. There was a rough preservation of the lobular liver architecture even in the absence of hepatic cells. About the foci of hepatic cells there was compression of the connective tissue. The liver cells showed changes consistent with a mild hepatitis (see Fig. 6).

CONCLUSION

The epidemiologic and clinical features of infectious hepatitis and homologous serum hepatitis were presented in this paper. Three cases are reported to illustrate the clinical variations and the basic pathologic changes. The following pathologic findings characterize infectious hepatitis and display variations only in degree and extent of involvement: (1) hepatocellular necrosis, central or generalized, with collapse of the reticular framework; (2) infiltration by monocytes, plasma cells, and lymphocytes, most marked in the portal areas; (3) varying degrees of regeneration, chiefly from remaining liver cells; (4) bile duct proliferation.

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PAROXYSMAL HEMOGLOBINURIA AS A RESULT OF THE COLD HEMOLYSIN*†

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The pathologic physiology of hemoglobinuria, irrespective of the initiating morbid process, consists of:

1. Hemolysis of erythrocytes at such a rate that the body is unable to metabolize the excessive hemoglobin pigments and hemoglobinemia results.
2. The glomerular filtration rate for hemoglobin exceeds the rate of tubular absorption (the renal threshold).

The wide range of clinical conditions exhibiting hemoglobinuria at times may be seen in the following classification from Kracke (7)

I. *Hemoglobinuria*
  A. Exogenous causes:
    1. Chemical agents
    2. Febrile intoxications
    3. Parasites
      a. Malaria
      b. Oroya Fever
    4. Transfusions
    5. Burns
    6. Cobra and spider venom
    7. Bay sickness
  B. Endogenous causes:
    1. Familial hemolytic anemia in severe relapse
    2. Atypical hemolytic anemias
    3. Acute hemolytic anemia of Lederer
    4. Cooley's anemia of childhood and infancy
    5. Sickle cell anemia

II. *Paroxysmal Hemoglobinuria*
  1. Cold hemoglobinuria (e frigor)
  2. March hemoglobinuria
  3. Nocturnal hemoglobinuria (Marchiafava—Micheli Syndrome)
  4. Allergic hemoglobinuria (Favism)
  5. Paroxysmal myoglobinuria (?)

In 1929 McKenzie (10) very ably summarized the subject of paroxysmal

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hemoglobinuria and reviewed the literature. Because of the absence of positive data in the many early case reports, it is impossible to cite any one author for its discovery. Dressler, 1854, (10), Elliotson, 1831, (10) and Stewart, 1794, (10) all cited cases of intermittent hemoglobinuria, but in all cure was effected with quinine, so they may have been caused by malaria. In 1880 Rosenbach (10) described the artificial production of hemoglobinuria by immersion of patients' feet in ice water for ten minutes. This test now bears his name. In 1904 Donath and Landsteiner explained the underlying mechanism of the disease and demonstrated a serologic test by which it may be recognized. They demonstrated that (a) the serum and plasma of these patients contains a lysin having the peculiar characteristics of being able to unite with red blood cells only at low temperature; (b) that the hemolytic reaction takes place in two phases—first, union of lysin and red blood cells at low temperature, and, secondly, after the mixture is warmed, lysis of red blood cells; (c) complement is essential for the second phase of the reaction; (d) the lysin is both an autohemolysin and an iso-hemolysin; (e) the red blood cells of these patients are not hemolyzed by the serum or plasma of normal persons. These serologic phenomena associated with paroxysmal hemoglobinuria as a result of cold are quantitatively constant.

The symptomatology of this disease involves not only the clinical events occurring during an attack, but also the chronic effects of blood loss and almost any of the complex pictures seen in late syphilis, whether acquired or congenital. Approximately 30 per cent reveal some stigmata of syphilis and 95 per cent or more have a positive serologic test for syphilis. Some authors feel that the congenital syphilitic is more likely to develop hemoglobinuria than is the acquired.

The first abnormality noted by the patient is usually the passage of dark red or black urine, which he usually refers to as being bloody. The interval between chilling and symptoms varies from a few minutes to several hours. Frequently the patient complains of such prodromal symptoms as aching pains in the back and legs or in the abdomen, abdominal cramps, headaches, and malaise.

The actual paroxysm usually begins with a shaking chill and a subsequent fever, but some cases have occurred without chill or fever. In the typical attack the first urine passed after the chill is highly colored, port wine, brownish chocolate, or black depending on the relative amounts of oxyhemoglobin, methemaglobin, and hematin present. Generally the hemoglobinuria is confined to the first one or two specimens after the paroxysm, but occasionally lasts two or three days. Spectroscopically the two absorption bands of oxyhemoglobin are seen. Chemical tests for hemoglobin are positive and microscopically red blood cells are either absent or few in number and chiefly "ghost" cells. Slight icterus for a few days may follow severe paroxysms, and splenomegaly during an attack is not uncommon. Hepatomegaly is occasionally seen.

There is a small group of patients who have paroxysmal hemoglobinuria as a result of the cold hemolysin but have complete absence of constitutional symptoms.

The degree of chilling required to induce an attack is extremely variable ranging from sub-freezing to spontaneous attacks during the summer. The Rosenbach test will nearly always induce hemolysis.

An interesting group of vasomotor phenomena has frequently been observed. Cases have been described which, following exposure to cold, developed urticarial lesions on face and body prior to the onset of hemoglobinuria. At times the lesions occur during the paroxysm. Raynaud's syndrome has frequently been observed both in the interval between chilling and the onset of the paroxysm and also during the acute paroxysm.

Witts (13) ably summarized the paroxysmal hemoglobinurias and concluded: "A feature of the paroxysms which has not attracted the attention it deserves is the occurrence of vascular disturbances which may threaten the life and comfort of the patient almost as seriously as the anaemia or the suppression of urine. They suggest that the haemolysis is preceded by agglutination of the red blood cells, or is accompanied by damage to the endothelial lining of the vessels, with a consequent tendency to thrombosis and blockage. Lesions of the same type have been produced in animals by injection of hemolysin. The vascular disturbances which occur in association with the paroxysms of hemoglobinuria e frigor are often described as Raynaud's syndrome, but they may be followed by gangrene of the tips of the ears, fingers, toes or nose. Gangrene never results from spasm alone, and in such cases an organic obstruction must be present. The records of the acute hemolytic anaemia of Lederer contain accounts of numb and painful fingers with red node-like spots on the hands; infarction of the spleen; abdominal pains followed by haemorrhage from the bowel; and patchy consolidation of the lungs. At autopsy multiple thromboses have been found in the arterioles, capillaries, and veins. The vascular complications observed in Marchiafava—Micheli disease include intracranial sinus and retinal thromboses, mesenteric thromboses, portal thrombosis, pulmonary infarction, and thromboses of the veins of the limbs. We are probably justified in concluding that the dangers common to all forms of intravascular hemolysis and hemoglobinuria are shock, anaemia, anuria and thrombosis."

The diagnosis of paroxysmal hemoglobinuria as a result of cold is easy in the average case; the history of the sudden appearance of "bloody" urine following exposure to cold in a syphilitic patient, the ability to reproduce artificially a paroxysm by the Rosenbach test, and a positive Donath-Landsteiner test. Unfortunately there is a group of patients who have minimal or no constitutional symptoms, but are referred because of anemia or intermittent albuminuria, which present the diagnostic problem. In these patients, particularly those with syphilis, one must bear in mind the possible relationship to paroxysmal hemoglobinuria and specifically look for hemoglobin in the urine and the presence of the cold hemolysin. There are numbers of case reports of cures in this type of paroxysmal hemoglobinuria by the successful treatment of the underlying syphilis.

CASE REPORT

W. W., a 31 year old negro soldier, a native of Louisiana, was admitted to a U. S. Army General Hospital in England, August 24, 1944, because of nervousness and shaking of the head. Both parents were dead and there were no other siblings; familial history was otherwise unknown to the patient. He had the usual childhood diseases without complications. In 1935 he began to experience frontal headaches which gradually increased in severity so that he developed insomnia. His history of this illness was vague as to any other symptomatology. After one month of illness he awakened one morning feeling drowsy and did not go to work. Approximately two hours afterwards he was found unconscious and was taken to Charity Hospital in New Orleans. He remained there three months but had no recollection of anything during the first two months. He stated that he was told he had "malarial fever of the brain." After discharge he was followed in the out patient department where he received injections in his veins and hip for reasons unknown to him. He denied having had a chancre. He stated that two lumbar punctures were done during his period of unconsciousness and one six months later but he did not know the results. He was re-admitted to Charity Hospital in 1937 because of a scalp laceration and was again given arm and hip treatments for three months. During this year he contracted gonorrhea.

In October 1943 this soldier was inducted into the Army, at which time his blood and spinal fluid were examined, and he was started on anti-syphilitic treatment in November 1943. He received twenty doses of Mepharsen and eleven of Bismuth, according to his "Supplementary Record."

About August 1st, 1944 this soldier was admitted to another U. S. army hospital because of nervousness and "twitching" of his head. While there two lumbar punctures were done which were "negative" and he was returned to duty. His commanding officer immediately had him admitted to the hospital.

His complaints on admission were nervousness and twitchings of his head which had been present for nine years.

Review of systems revealed a life-long history of recurrent episodes of dark urine varying from dark amber to black following exposure to cold. There were no constitutional symptoms associated with these episodes.

The physical examination revealed an apprehensive, well developed and nourished Negro with bizarre, irregular twitchings of his head which were intermittent and disappeared when his attention was actually engaged. Skin was of normal texture and without icterus or demonstrable lesions. His ears, eyes, nose, mouth and pharynx presented no abnormalities. The neck revealed no enlargement of thyroid or adenopathy. The lungs were clear. The heart was of normal size and shape, without murmurs. The rhythm was sinus and the rate was 80 per minute. The blood pressure was 118 systolic over 78 diastolic. The abdomen was muscular and had no masses or tenderness. The liver, kidneys and spleen were not felt. There were no herniae. The genitalia were normal. A rectal examination revealed no hemorrhoids, there was good sphincter tone, and a slightly enlarged prostate of normal consistency. The skeletal system was not unusual. A neurologic examination revealed physiologic deep reflexes and no pathologic ones. There was bizarre intermittent jerking of the head except when attention was diverted.

Laboratory findings:

A. *Blood:* Hemoglobin varied 75 per cent–85 per cent; Red Blood Cells varied 3,500,000 to 4,600,000; Leucocytes varied 5,400–8,200; normal differential. Thick smear for malarial parasites—negative.

B. *Serology:* Kahn positive on six occasions. 3 to 4 Kahn units. Wassermann positive on two occasions and anti-complementary once.
Donath-Landsteiner positive on three occasions, approximately 10 per cent hemolysis.

C. *Blood Chemistry:*
Sugar, 108 mgm. %
N. P. N., 16.6 mgm. %
Icteric index, 4
Van den Berg—Direct—0
Indirect—Less than 0.25 mgm.

D. *Urinalysis:*
Straw color; 1.023; acid; albumin—negative; sugar—negative; microscopic—negative.

The patient was exposed to the weather at a temperature of 60° F. for 20 minutes and when returned to the hospital ward voided port wine colored urine which contained three plus albumin and gave a positive chemical test for hemoglobin; the sediment contained no red blood cells.

Since this patient had been treated for syphilis on three separate occasions with arsenicals and bismuth and still had positive serologic tests for syphilis, although of low titre, and still had cold hemolysin manifested by positive Donath-Landsteiner it was decided to try penicillin. He was therefore given 4,800,000 units of penicillin over a period of fifteen days. At the end of this course of therapy the Kahn titre and the Donath-Landsteiner titre were unchanged.

SUMMARY

1. A 31 year old negro soldier with paroxysmal hemoglobinuria as a result of the cold hemolysin is presented. He has been unsuccessfully treated for syphilis with arsenicals and bismuth on three occasions.

2. Penicillin therapy consisting of 4,800,000 units over fifteen days failed to influence either the titre of Kahn reaction or of the Donath-Landsteiner reaction.

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ADVANCES IN ANESTHESIA*†

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The practice of anesthesiology has advanced so rapidly that it is difficult for the surgeon and average anesthetist to keep informed as to the progress made in this specialty. Anesthesiology is constantly being affected by the introduction of new agents and technics, by re-evaluation of older methods, and by narrowing or widening the range of usefulness of various technics. In addition to the anesthetist's principal functions as an important member of the operating team, he is able to apply his skill toward the relief of intractable pain which cannot otherwise be relieved safely and effectively, toward furnishing valuable diagnostic information, and in treating favorably certain vascular disturbances.

It is the purpose of this paper, therefore, to discuss some of the newer advances in the field so that the general surgeon may become familiar with them and utilize the services of the professional anesthesiologist to the best of his advantages in caring for surgical patients.

INHALATION ANESTHESIA

Inhalation anesthesia has had no important radical innovations in recent years. Progress has, however, been made toward a better understanding of the agents used and a wider application of their uses by improvement of technic.

Diethyl ether is still the drug most widely used for inhalation anesthesia. The preferred method of administering ether is by means of a modern gas machine using the closed carbon dioxide absorption technic. However, such equipment is not available in a majority of the smaller hospitals, so that the open drop technic is employed because of its simplicity and safety in the hands of the inexperienced. Admittedly, the resulting anesthesia may be far from ideal, but from the point of view of safety to the patient and adequate anesthesia, no other agent is as valuable as ether in the absence of a trained anesthetist or suitable apparatus. The use of 100 per cent oxygen throughout the course of anesthesia will help greatly to decrease the postoperative morbidity resulting from severe anoxia which occurs with this form and method of anesthesia. The maintenance of a patent airway and the postoperative use of oxygen and carbon dioxide will facilitate recovery from anesthesia and tend to prevent pulmonary complications.

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*From the Department of Anesthesiology.

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Chloroform. The disadvantages of chloroform outweigh its advantages and its use should be condemned. The dangers of this drug are not only immediate but may occur as long as one month after administration.

Divinyl ether or vinethene is a newer volatile agent and is administered by the open drop method. It is an excellent drug for short surgical procedures, lasting up to thirty minutes, or as an induction agent preceding diethyl ether anesthesia, especially in children. Induction time is approximately one to three minutes and the patient is usually awake within five minutes after cessation and in full control of all his faculties. Nausea and emesis are rarely encountered, but preoperative atropine should not be omitted because of increased salivation. Renal or hepatic disease is a definite contra-indication to its use.

Ethyl chloride. The inherent toxicity and rapidity of induction should make one pause before using it for a general anesthetic. It is toxic to the myocardium and in the presence of excessive epinephrine output, will produce, especially during the induction stage, a ventricular fibrillation from which one is not likely to recover. Ethyl chloride should be displaced for the greater safety of divinyl ether.

Metapryl. One of the newest volatile anesthetics is N-propyl methyl ether or metapryl developed by Dr. John Krantz at the School of Medicine of the University of Maryland. It has passed the experimental stage and is well advanced into the clinical phase. This anesthetic shows great promise; however, its use is still limited to larger clinics and hospitals with well organized departments of anesthesiology.

GASEOUS INHALATION AGENTS

There are three gaseous inhalation agents at the disposal of the anesthesiologist.

Cyclopropane is the newest and probably the most ideal as it provides a pleasant and rapid induction. It is very useful as a supplement to spinal and local anesthesia, and as the sole agent of extra-abdomoninal surgery. It also provides adequate relaxation for the majority of intra-abdominal operations below the umbilicus. Cyclopropane should be administered only by an experienced anesthesiologist who can evaluate its characteristics and can interpret the circulatory phenomena which occur with this gas. Its property of producing cardiac arrythymias cannot be regarded lightly, especially in thyrotoxicosis; however, its use in this disease is not definitely contra-indicated. The administration of this gas is probably not a wise choice when there is a history of cardiac irregularity. A sympathomimetic agent should never be used in the presence of cyclopropane. For patients in shock, cyclopropane is probably the anesthetic of choice, for too frequently patients go into marked surgical and anesthetic depression following the use of ether anesthesia.

Ethylene has been widely employed as an anesthetic agent for many years, but probably not to the extent which its properties merit. When properly used, ethylene disturbs the normal physiology of the body very little. The technic of administering this gas is similar to that for nitrous oxide, but its slightly greater potency permits a somewhat higher percentage of oxygen in the anesthetic mixture. However, the explosive properties of ethylene are comparable to those of cyclopropane and ether-oxygen anesthesia.

Nitrous oxide may be used with safety when a general anesthetic agent of low potency is indicated if adequate oxygen is included in the mixture. Neglect of this cardinal principle is the factor which leads to trouble with nitrous oxide. Those who claim that anesthesia with this agent is produced by its asphyxial effects are ignorant of the anesthetic properties of this gas and its correct administration. Adequate oxygenation must be maintained at all times, and, if the patient has been properly premedicated with the barbiturates and morphine-scopolamine before surgery, this may be accomplished. Satisfactory nitrous oxide-oxygen anesthesia requires the services of an anesthesiologist who can promptly interpret the signs of oxygen lack or anoxia if a fatality is to be averted. Wisdom often will indicate the use of a different agent in the vigorous, unpremedicated or aged patient. Practically, the most important property of nitrous oxide is the fact that in mixtures with oxygen it is non-explosive.

INTRAVENOUS ANESTHESIA

Intravenous anesthesia with *sodium pentothal* is finding increasing usefulness, especially with the impetus given to the method through its frequency of use by military surgeons. Sodium pentothal should not be used as the sole anesthetic agent in operations lasting more than one hour. Marked relaxation may be obtained with the drug if a deep anesthesia is produced, but this is not considered safe and the ever-present hazards of respiratory depression and laryngospasm should be kept in mind. Equipment to furnish artificial respiration with 100 per cent oxygen, under pressure if necessary, and instruments for endotracheal intubation, should be immediately available. Other safety precautions are the use of a dilute solution (2.5 per cent) to minimize the dangers of overdosage, phlebitis and tissue damage as a result of extravenous injection, and the realization that renal or hepatic insufficiency contra-indicate the use of sodium pentothal.

Primarily, pentothal is employed to produce anesthesia for short surgical procedures, but is finding a wider range of usefulness. It serves: to supplement spinal anesthesia, as an induction agent, as a basal rectal anesthetic, as a supplement to local, regional, nitrous oxide or splanchnic block anesthesia, as an anesthetic for diabetic patients, and to arrest convulsions.

The intravenous anesthetic agents require proper medication with atropine before hand to prevent vagal and sympathetic stimulation which may occur with these agents.

RECTAL ANESTHESIA

The most frequently used rectal anesthetic is *avertin* or tribromethanol with amylene hydrate, it is used as a basal anesthetic and is very satisfactory for the apprenhensive, overactive or excitable patient. Any of the accepted inhalation anesthetics will complete the anesthesia. One must constantly watch for postoperative depression, and hospitals which are understaffed with nurses should not use this procedure as patients arousing from avertin anesthesia require constant observation.

REGIONAL ANESTHESIA

Spinal anesthesia is still considered as being too hazardous by some surgeons. The hazards of spinal anesthesia are caused by faulty administration of the agent and very infrequently to the drug itself. When a spinal anesthetic agent is administered and then a nurse is left in charge of the patient, surgeons are negligent in their duties. Spinal anesthesia is a safe procedure in the hands of the trained anesthesiologist who understands its characteristics and is equipped to combat untoward disturbances of physiology, especially vascular hypotension and respiratory insufficiency. The circulatory disturbance usually is controlled readily by vasopressor drugs, and by intravenous solutions, plasma or blood. Respiratory difficulty, usually caused by the presence of the anesthetic drug cephalad to the fourth or fifth thoracic segment, is combated readily by artificial respiration with a gas machine. Respiratory failure or embarrassment resulting from spinal anesthesia usually is transitory but can be fatal in the absence of adequate aid from the anesthetist.

The proportion of cases in which spinal anesthesia is used is now greater than ever before. At the University of Maryland Hospital it is employed for almost every laparotomy as well as for a goodly portion of operations on the pelvic organs when approached from below, on the perineum, and on the lower extremities. For intra-abdominal operations it is denied only to patients so extremely sick that they appear to be able to tolerate nothing but field block or local infiltration with or without light gas anesthesia. There are several reasons why spinal anesthesia is so frequently used. The quantities of the anesthetics needed are so minute that the functions of kidneys, liver and blood are little disturbed. This is in marked contrast to the pronounced changes that occur during and following the depths of ether anesthesia necessary to give relaxation approaching that produced by spinal anesthesia. This relaxation permits the surgeon to operate with a minimum of trauma and the least spreading of infection. Thus, in the presence of distention or of peritonitis, spinal anesthesia, if carefully administered and watched, saves many patients from surgical death. Postoperative malaise is much less following spinal anesthesia than following comparably deep ether anesthesia. The newer drugs produce anesthesia of sufficient length for almost all operations below the diaphragm. Serious objections which have been raised to spinal anesthesia are not always valid when proper technics are employed.

Continuous or fractional spinal anesthesia is satisfying a long felt need in furnishing the desirable features of spinal anesthesia over periods of several hours. Proper levels of anesthesia can be obtained consistently, by the fractional method with the administration of small amounts of the drug. It will also allow the surgeon to experience the same degree of relaxation at the end of the operation as at the beginning without the time factor being so important.

Continuous caudal anesthesia, which has been made possible by adapting the malleable needle used for fractional spinal anesthesia or by the insertion of a ureteral catheter into the sacral hiatus, seems to be answering a real need for improved obstetric anesthesia. In several years, after this method has been given a critical trial in institutions throughout the country, a worth while evaluation of its place in obstetrics will be forthcoming. Caudal anesthesia is not only useful in obstetric procedures, but it has been found to have a definite place in general surgery, especially in operations around the perineal regions. A word of caution is necessary at this time: the administration requires the services of a well-trained anesthesiologist and the use of special equipment. For this reason continuous caudal anesthesia will not be available to all obstetric patients.

Regional nerve block anesthesia is especially indicated in feeble patients and patients in shock. The technics for producing this type of anesthesia for operations on all parts of the body have been quite well standardized and described in the literature. The length of time needed to carry out this type of procedure and the frequency of unsatisfactory results, especially in the hands of the novice, have restricted its use. Furthermore, marked dexterity on the part of the surgeon is required as well as the services of a trained anesthesiologist. The anesthesiologist particularly must be able to impart to the patient the feeling that he will not experience any discomfort, while the surgeon must respect the tissues and avoid any unnecessary traction or packing.

The technics of regional sensory nerve block employing absolute alcohol are recommended to relieve intractable pain from cancer and similar conditions when other measures prove futile or undesirable.

When pain of sympathetic origin or causalgia is suspected, blocking the sympathetic nerve supply to the painful area with a local anesthetic drug is of diagnostic value, and will indicate whether surgical intervention is needed. This procedure is also valuable in determing the probable value of sympathectomy in peripheral vascular disease. In the acute stages of thrombophlebitis the same type of block is a beneficial therapeutic measure.

Refrigeration anesthesia or crymotherapy is useful for amputations especially in the poor risk patient. It is produced by packing an extremity in ice or placing it in a special refrigeration apparatus following the application of a tourniquet to stop all flow of blood in the limb. No supplemental anesthesia is necessary and the incidence of shock and infection is said to be reduced. Postoperative pain and sepsis are diminished by maintaining a low tissue temperature postoperatively. Refrigeration anesthesia is also recommended as temporary treatment for badly traumatized extremities until adequate surgical facilities are available. Shock resulting from pain, trauma and hemorrhage are less severe and bacterial growth is inhibited by the cooling.

The utilization of *curare* is at the present moment a matter of foremost interest in anesthesiology. Its value has been made evident by numerous writers. Curare can be used in conjunction with all the common, general anesthetic agents; however, its administration will vary according to the agent employed.

Accumulated experience has forced the adoption of one fundamental rule of technic while using curare. It is simply; the patient is kept breathing. The administration of curare is a relatively safe procedure, possibly safer in comparison with other major anesthetic technics, provided this seemingly absurd, simple rule is not violated. Compliance with this admonition is not the easy matter it would at first appear. The anesthetist frequently encounters variations in response to the effect of the drug, depending on the age, general physical condition, the type of surgery being performed, the anesthetic agent being used and the individual susceptibility to the drug. Besides this, either over-confidence or unfamiliarity with the drug lead to the occasional misjudgement of dosage, resulting in apnoea and the necessity for artificial respiration or resuscitation. The proper dose, as with other drugs, is the smallest dose which will give the desired effect.

OXYGEN AND HELIUM THERAPY

A field of anesthesia in which the average surgeon does not expect the anesthesiologist to be interested in that of oxygen and gas therapy. The well trained anesthesiologist assists the surgeon in the prevention of cerebral and cardiac anoxia by the proper utilization of oxygen therapy. The anesthesiologist is constantly on the alert for signs of impending anoxia and if the condition is recognized early, postoperative morbidity will be decreased. Anoxia may manifest itself in various ways either upon the nervous, circulatory or respiratory system. Oxygen therapy is also beneficial in the prevention of postoperative distension. The subcutaneous use of oxygen in the treatment of angina pectoris and coronary heart disease has also been found of great value by physicians in the field of anesthesiology.

Helium oxygen mixtures are particularly beneficial to the asthmatic patient as well as in the treatment of infections around the neck where laryngeal obstruction is present. The utilization of this mixture will frequently tide the patient over the critical periods until surgical intervention is possible.

It is hoped that from the foregoing paragraphs the surgeon and physician may obtain a brief summary of the present status in the field of anesthetic agents and technics. It is further hoped that a more widespread recognition of their relative values may be obtained with the ultimate idea of benefiting both the surgeon and the patient.

THE USE OF RADIUM IN THE TREATMENT OF ADENOID TISSUE*†

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The otolaryngologist and the pediatrician are frequently faced with a child who has infected adenoids or sinus suppuration secondary to an obstructive adenoid mass. The child may be quite young, under two years of age, and there may be some infection of the tracheobronchial tree as well. Should one do a surgical adenoidectomy, or should radium be applied to the nasopharynx? This is often a moot question, perhaps more because there is widespread lack of appreciation of the relative value of these procedures rather than because of any real question as to their individual merits.

Nasopharyngeal radiation is not a substitute for surgical adenoidectomy; rather, the two procedures are therapeutic adjuncts. Each has its definite indications, and limitations, and not in-frequently both are best used together.

RADIATION THERAPY (1)

Radium element itself is therapeutically inactive. However, there is a constant emanation of three rays (alpha, beta and gamma) from radium, and these rays are active therapeutically. This mixture of emanations is known as "radon". In practice, one of two methods may be employed to make these rays available for therapy. In the first, radium element is placed in a sealed capsule, and after a period of days an equilibrium is established betweeen the rate of emanation of the rays from the radium element and the rate of loss of these rays through the walls of the capsule. When this state is reached, a constant source of rays is available, the therapeutic dosage of which can be accurately calculated. In the second method, radon rays are captured from a large storage supply of radium and the rays are placed in sealed capsules (radon capsules). Since there is no replacement within the capsule of the rays passing through the capsule wall, the radon content of these capsules is exhausted in a matter of hours. Therapeutically, it makes no difference which type of capsule is employed, so long as the dosage is accurately calculated.

The *alpha rays* are positively charged particles travelling at a rate of from 12,000 to 18,000 miles per second. They are completely absorbed by

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three inches of air or by a thin screen (even a sheet of writing paper). These rays are of little value for therapy as they have no penetrating ability. Practically, they are of little concern to us, as all commercially available capsules completely filter out these rays.

The *beta rays* are simple negative electrons, travelling at from 60,000 to 180,000 miles per second. These rays are one-half absorbed in 1 mm. of soft tissue, and practically entirely absorbed by 3 mm. of soft tissue. They are also completely absorbed by 1 mm. of lead or 4 mm. of aluminum. Beta rays are produced in far greater numbers than gamma rays, and have similar therapeutic effects. However, there is a greater tendency toward a burn effect, and short exposures are therapeutic.

The *gamma rays* are high frequency ether vibrations similar to light, and travel at the rate of 186,000 miles per second. Gamma rays have the capacity to penetrate 6 to 8 inches of soft tissue. They are practically completely absorbed by 5½ inches of lead. These rays have generally been considered best for therapy because of their ability to penetrate soft tissues and their relative freedom from superficial burning. Long exposures are usually necessary for therapeutic dosage, and a suitable capsule must be chosen which will filter out the more irritating beta rays.

Deep roentgen-ray therapy has similar effects to gamma radiation but affects a larger area of tissue and is not devoid of secondary effects on the tissues and organs penetrated to reach the affected part. Therapeutically similar effects can be expected from either beta rays, gamma rays or deep roentgen-rays, in comparable dosage, and not infrequently better results can be obtained by their combined use.

Radiation therapy is directed at cellular growth and multiplication. Certain cells of the body are markedly affected by radiation, and the lymphoid tissue cells stand high in the list of "radio-sensitive" cells. When radiosensitive cells are exposed to such radiation, the cellular nuclei are affected; mitosis and cellular division are inhibited, and cellular growth diminishes. As the life cycle of the cell is completed, fewer replacement cells than usual are present, and the tissue growth appears smaller. In the usual therapeutic dosage, radiation does not destroy or remove lymphoid tissue. It affects the germinal center, and secondarily reduces its size. Irradiation has a similar powerful effect on the bacterial cells growing within this tissue. (2) This effect is not permanent.

RADIATION VS. SURGERY

Where the amount of lymphoid tissue present is large, and especially if the original mass has never been removed, it is usually better to remove the mass surgically. This is particularly true if immediate results are needed, since the effects of radiation are relatively slow of onset and only moderate in amount.

If the amount of tissue present is small, and the symptoms are caused by its strategic location (as around the tubal orifices with resultant conduction hearing loss or recurrent middle ear abscesses), then radiation therapy is usually the method of choice. Surgical means frequently fail to remove completely all lymphoid tissue from the nasopharynx, especially laterally, and here radiation is most effective. Frequently, at the time of adenotonsillectomy, the otolaryngologist realizes that he has been unable to remove the lymphoid tissue of the nasopharynx completely. Such patients should receive radiation therapy soon afterward.

In very young children, it is often wise to irradiate after surgery in an effort to prevent recurrence of the tissue. It is generally recognized that after adenoidectomy in children under age 2 years, there will be a recurrence of some tissue in almost 100 per cent of the cases. All of these will not require treatment, but a large percentage will require secondary surgery or radiation, or both, sooner or later. The recurrence rate in adenoidectomy after puberty is practically *nil,* and for practical purposes, it is unlikely after the age of 8 or 9 years. Ideally, then, adenotosillectomy would best be done at age 8 or 9 years instead of at age 4 to 5 years, as is now the custom. Unfortunately, most children in urban centers have developed real indications for tonsillectomy and adenoidectomy at an earlier age.

The problem is the same whether surgery or radiation has been employed. If the factors responsible for recurrence are present (infection, allergy, etc.), the recurrence rate will be the same whether surgery has been performed or radiation has been employed. On the other hand, radiation is most effective against this recurrent tissue. This is especially true in young people, as the younger the patient, the more radio-sensitive are the lymphoid tissue cells.

In short, surgery is best where immediate results are required and where large quantities of tissue are involved. Radiation, on the other hand, finds its greatest usefulness in the treatment of recurrent lymphoid tissue and in the eradication of lymphoid tissue which is inaccessible to surgery, such as that located laterally in the nasopharynx or in the tubal orifices

CHOICE OF RADIATION

The best results obtained by the author in the treatment of these cases by radiation has been from combined deep roentgen ray therapy and gamma radium radiation. The average case has received 1000 tissue roentgens to the nasopharynx within thirty days, given in five or six divided doses. At the time of the second and fifth treatments, 50 milligram hours of gamma radium radiation have been given to each side of the nasopharynx, making a total of 200 milligram hours in all. (In those under 7 or 8 years of age, a gamma radium applicator cannot be inserted through the nose, so that this method of treatment could not be employed). All such treatments were given by Dr. Howard H. Ashbury for the author.

Other cases have been treated by roentgen rays alone, also with good results. Either of these methods requires repeated visits to the roentgenologist and requires considerable time. They are, therefore, not expedient when treating a large series of cases. In addition, several annoying side effects invariably accompany the treatment; i.e., temporary hair loss from the face, and dryness of the mouth.

The use of *beta radium rays* alone for this therapy was first described by Crowe and Burnam (3), and represented an effort to expedite the treatment of large numbers of such cases. With this method the patient requires from one to four treatments (usually three) at two to three week intervals. Each treatment takes a total of from thirteen to seventeen minutes (six and a half to eight and a half minutes to each side of the nasopharynx), and the treatments are carried out by the otolaryngologist in the office or clinic. Rarely an acute otitis media follows such a treatment, but this can usually be obviated by avoiding exposure of the nasopharynx to radium during acute upper respiratory infections. No other sequele have been reported (2).

A special nasal applicator has been devised for using beta radiation, consisting of a small Monel metal capsule containing 50 mgm. radium, a brass wire stem, and a light-weight aluminum handle (4). The outside diameter of the radium capsule is only 2.3 mm., so that it can usually be passed, even through a very young infant's nose. Under local anesthesia (or without anesthesia), the applicator is passed along the floor of the nose and fixed in position with a strip of scotch tape. Both sides of the nasopharynx are radiated at the same sitting, and the treatment is best repeated in from fourteen to twenty one days. No visible results are manifest under four to six weeks, and usually longer is required. Some secondary injection of the nasopharynx will be observed for a few days after each treatment, but this soon subsides.

Although the results from this method have not been quite as good as with the combined use of deep roentgen ray and gamma radium radiation, the use of beta radium radiation alone has much to commend it. By and large excellent results are obtainable with this therapy in selected cases. In addition, its ease of administration make it the only suitable method for dealing with large numbers of cases (as in clinics), at the operating table, and in young infants.

Bordley (2) recently cautioned that, "The danger to the future of radium therapy is in its overenthusiastic use by otologists not willing to take the care and the time to study each individual case. The indiscriminate use of this technique may bring it into disrepute." This warning is wholeheartedly endorsed.

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DEPARTMENT OF OBSTETRICS

AN OBSTETRIC CASE REPORT*†

The 29 year old patient registered at the prenatal clinic on November 21, 1946. Her last menstrual period was on June 15, making the estimated date of confinement March 22. The serologic test for syphilis was negative, and the RH blood factor was positive. There was a history of one spontaneous miscarriage, which was uncomplicated. The external pelvic measurements were normal. *No internal mensuration* was done.

On admission, the prenatal history revealed that the patient had had some edema of the ankles for the past few weeks. She had been on a salt poor diet for the previous three months. The general physical examination was negative. The height of the fundus was 31 cm. above the symphysis. The estimated size of the fetus was about 9 pounds, and the presenting head was floating above the pelvic brim. The blood pressure was 130 systolic over 86 diastolic. Her pains had begun about one and a half hours prior to admission. Because the patient was essentially a primigravida in labor at term with the head floating, roentgen pelvimetry was done. The report was as follows: the AP diameter of the inlet 9.6 cm.; of the midpelvis 12.2 cm.; of the transverse diameter of the inlet 11.5 cm.; of the midpelvis 10 cm. and of the outlet 9 cm. There appeared to be a definite disproportion between the fetus and the pelvic inlet. After the patient had been typed and cross matched and blood obtained, a cesarean section was done. A full term, living child, weighing seven and a half pounds, was delivered by low cervical section. The patient's postoperative course was uneventful, and she was discharged from the hospital on her eighth postoperative day in good condition.

DISCUSSION

One cannot stress too strongly the importance of accurate information as to the size and shape of the pelvis in relation to the fetal head. It has been aptly said that the physician who neglects pelvimetry is comparable to one who attempts to treat pulmonary diseases without the aid of auscultation and percussion. External measurements are too frequently inaccurate indices of the size of the pelvic cavity. As early as 1866 Hodge told us that the value of the external measurements is only relative. Even today most physicians are not skillful in taking internal measurements, and as a result, there is a great tendency to neglect pelvic mensuration. The so-called test of labor, in which a cesarean section is performed if the head has not descended after a certain length of time, is, in most instances, not the most intelligent procedure. This method of handling a case of possible disproportion not infrequently costs the life of the mother or the baby, and sometimes of both. Accurate information as to the size and shape of the pelvis and of the fetal head is essential. Thomas, Jarcho, and others have done much to increase our knowledge in the field of roentgen ray pelvic mensuration; and

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* From the Department of Obstetrics, School of Medicine, University of Maryland.

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even more accurate measurements have been made available through the contributions in stereoroentgenometry by such men as Caldwell, Moloy, D'Esopo and others.

In the case under discussion the conflicting evidence of the reported normal extenal measurements and the contraction of the pelvic cavity as demonstrated by roentgenography is an example which calls to attention the danger involved in too great reliance on external pelvimetry alone.

ANNUAL REPORT OF THE DEPARTMENT OF OBSTETRICS, 1946

UNIVERSITY OF MARYLAND, SCHOOL OF MEDICINE

FOREWORD

Once again it is my pleasant duty to express my thanks to all members of the department for their efforts and for their cooperation, without which the work represented in this report could not have been done.

The number of patients cared for during the year increased by 373 as compared with 1945. The majority of this increase was in home deliveries, a matter of necessity rather than of choice, since the number of available hospital beds fell far below the demand and the overflow had to be accommodated.

Of the 5 maternal deaths during the year, 3 occurred in unregistered clinic patients. On the basis of 210 such patients treated, the mortality was 1.45 per cent. When this is compared to 2 maternal deaths in 1792 registered clinic patients, 0.11 per cent or $\frac{1}{13}$ of the rate in unregistered, the value of routine prenatal care is again demonstrated. Two of the 3 deaths in unregistered patients were caused by eclampsia and 1 as a result of rupture of the uterus. With proper care eclampsia should not occur and these 2 deaths were unnecessary. The patient whose uterus ruptured was admitted to the hospital after many hours of labor in her home. Undoubtedly the seriousness of her condition should have been recognized sooner.

A fetal mortality rate (uncorrected) of 5.54 per cent is entirely too high. We can say in this regard only that we are earnestly striving to lower this rate and hope to present better figures in the future.

Lewis H. Douglass

ANNUAL REPORT

1946

Number of patients delivered and discharged (not including abortions)		2685
Twins 37 sets		
White—twins 9 sets	1247	
Colored—twins 28 sets	1438	
In homes—twins 7½ sets		651—24.25%
White	11	
Colored—twins 7½ sets	640	
Registered		584
Unregistered		67
In hospital—twins 29½ sets		2035—75.75%
White—twins 9 sets	1236	
Colored—twins 20½ sets	799	

Number of deliveries—twins 37 sets	2722
White	1255
Colored	1467
Normal spontaneous	1082—39.676%
White	160
Colored	922
Operative deliveries—all in hospital	1640—60.324%
White	1095
Colored	545
Forceps	1477—54.28%
White	999
Colored	478
High	0
Mid	11
Presentation occiput posterior	3
As such	1
Following forceps rotation	2
Presentation occiput transverse	2
Following forceps rotation	2
Disproportion—cephalo pelvic	2
Mid-pelvis	1
Outlet	1
Twin-second	2
Rheumatic heart disease severe	1
Presentation brow	1
Low	1466
Control	1034
Presentation occiput posterior	131
As such	48
Delivered following rotation	83
Spontaneous	1
Manual	48
Forceps	34
Presentation occiput transverse	226
Delivered following rotation	226
Spontaneous	1
Manual	78
Forceps	147
Labor prolonged	72
Presentation face	3
Breech extraction	78—2.866%
White	45
Colored	33
Internal podalic version and breech extraction	8—0.29%
White	3
Colored	5
Presentation transverse	5
Twin-second	2
Placenta previa partialis	1

Craniotomy		3—0.11%
White	0	
Colored	3	
Disproportion cephalo pelvic labor prolonged—dead fetus	1	
Disproportion cephalo pelvic-outlet; deep transverse arrest-dead fetus	1	
Disproportion cephalo pelvic; post eclampsia dead fetus	1	
Operation destructive		1—0.038%
White	0	
Colored	1	
Disproportion cephalo pelvic-outlet; presentation transverse-shoulder impacted	1	
Cesarean section		72—2.64%
White	48	
Colored	24	
Classical	23	
Preeclampsia-moderately severe	1	
Preeclampsia severe	2	
Section previous-disproportion cephalo pelvic	3	
Rh negative—high titre antibodies	1	
Rh negative—no antibodies	1	
Section previous	3	
Cervix—carcinoma of	1	
Fibroids—uterine	1	
Obesity with uterine atony	1	
Placenta previa centralis	1	
Disproportion cephalo pelvic	4	
Syndrome, dystocia dystrophy	1	
Uterus—bicornute-presentation transverse	1	
Post eclampsia	1	
Cardiovascular renal disease—presentation transverse	1	
Laparotrachelotomy	48	
Disproportion cephalo pelvic	28	
Placenta previa	4	
Centralis	2	
Partialis	1	
Marginalis	1	
Obstetric history poor: postmaturity	2	
Diabetes mellitus	2	
Stillbirths—repeated	1	
Preeclampsia	2	
Rh negative with high titre	1	
Rh negative with antibodies	1	
Elderly primigravida with breech	1	
Pelvis contracted—absolute	1	

Section previous—placenta under scar	1		
Hypertensive cardiovascular disease	1		
Presentation face	1		
Disproportion cephalo-pelvic—due to tumor in pelvis	1		
Placenta abruptio	1		
Section postmortem		1	
Eclampsia fulminating	1		
Laparotomy			1—0.038%
Uterus—rupture of		1	

Abortions					84
Spontaneous				81	
In homes			24		
Registered		13			
Cause unknown	13				
Unregistered		11			
Syphilis—maternal—untreated	3				
Cause unknown	8				
In hospital			57		
Cause unknown	54				
Fibroids uterine	1				
Hyperemesis gravidarum—severe	1				
Disease—pelvic inflammatory	1				
Therapeutic				3	
Hypertensive cardiovascular disease severe	1				
Preeclampsia fulminating	1				
Monster—anencephalic	1				

Other operations and procedures not including delivery				2431
Episiotomy			1434	
Central		1395		
Others		39		
Perineorrhaphy			190	
Indicated		128		
Elective		62		
Trachelorrhaphy			165	
Indicated		82		
Elective		83		
Proctoperineorrhaphy			54	
Hysterostomatomy			21	
Vaginal mucous membrane—laceration of—repair of			78	
Hysterectomy			3	
Pan		1		
Cervix—malignancy of	1			
Supravaginal		2		
Fibroids—uterine	1			
Uterus—rupture of	1			
Dilatation and curettage			4	
Secundines retained		2		
Mole—hydatidiform		2		

Placenta adherent—manual removal of				11	
Placenta retained—manual removal of				5	
Erosion cervical—cauterization of—with silver nitrate				25	
Fetal scalp clamp—application of				17	
Head—aftercoming—forceps to				25	
Head—manual rotation of				128	
Head—forceps rotation of				184	
Maneuver—Mariceau—Smellie—Veit				4	
Cyst—Bartholin—excision of				1	
Polyp—cervical—removal of				1	
Oophorectomy				1	
Colporrhaphy anterior				1	
Veins—superficial—ligation of				1	
Condyloma acuminata—excision of				1	
Amniorrhexis for induction of labor				16	
Preeclampsia severe			10		
Rh negative with antibodies			2		
Hypertensive cardiovascular disease severe			1		
Placenta previa—marginalis			2		
Monster anencephalic			1		
Sterilization Madlener				1	
Section previous			1		
Sterilization Pomeroy				60	
Section previous			8		
Multiparity			43		
Deficiency mental			1		
Obesity			1		
Hypertensive cardiovascular disease—severe			1		
Rheumatic cardiovascular disease—severe			2		
Sclerosis multiple			1		
Colitis—ulcerative—severe			1		
Pelvis—infantile—also with paralysis of lower limbs			1		
Syndrome—dystocia dystrophy			1		
Number of patients discharged from hospital					2452
Admitted from prenatal clinic				1219	
White	294				
Colored	925				
Reasons for admission:					
Normal			718		
Toxemia			82		
Preeclampsia		48			
Severe	16				
Mild	32				
Hypertensive cardiovascular disease		27			
Mild	16				
Moderately severe	7				
Severe	4				
Eclampsia		2			
Antepartum	2				
Hyperemesis gravidarum		5			

Abortion		23
Threatened	3	
Complete	20	
Hemorrhage vaginal		49
Antepartum	47	
Postpartum	2	
Hematuria—cause undetermined		2
Labor false		29
Labor premature		31
Multiparity		33
Presentation breech		20
Placenta—premature separation of		10
Placenta—retained		3
Pregnancy multiple		17
Membranes—premature rupture of		77
Sections previous		8
Retention urinary		3
Perineum—laceration of		20
Paralysis agitans		2
Disproportion—cephalopelvic		14
Vagina—laceration of		1
Observation		15
Rheumatic cardiovascular disease		8
Sclerosis multiple		3
Pyelitis		12
Antepartum	11	
Postpartum	1	
Secundines retained		4
Labor—arrested—second stage		1
Endometritis—puerperal		9
Fever—cause undetermined—postpartum		1
Hydramnios		3
Rh negative with high titre		1
Arthritis gonorrheal		1
Mastitis postpartum		3
Uterus—anomaly of—bicornute		1
Thrombophlebitis—legs		1
History obstetric—poor		1
Pelvis—contraction of—absolute		1
Abdomen—mass of		1
Uterus—fibroids of		1
Cystitis antepartum		2
Atrophy—muscular		1
Anemia secondary		1
Polyneuritis		1
Postmaturity		1
Cervix—carcinoma of		1
Malnutrition		1
Infection—upper respiratory		1
Pain—abdominal—obstruction intestinal		1

From other sources						1233
Private					1090	
Reasons for admission:						
Normal				743		
Abortion				45		
Threatened			12			
Complete			33			
Toxemia				38		
Preeclampsia			17			
Mild		10				
Moderately severe		3				
Severe		4				
Hypertensive cardiovascular disease			14			
Mild		9				
Moderately severe		1				
Severe		4				
Hyperemesis gravidarum			7			
Mild		3				
Severe		4				
Hemorrhage vaginal				21		
Antepartum			20			
Postpartum			1			
Labor false				55		
Labor premature				11		
Membranes—premature rupture of				78		
Disproportion cephalopelvic				14		
Presentation breech				21		
Pyelitis antepartum				10		
Glycosuria of pregnancy				1		
Observation				11		
Section previous				12		
Obstetric history—poor				1		
Rh negative with antibodies				4		
Pneumonitis				1		
Multiparity				1		
Cardiospasm				1		
Endometritis—postpartum				1		
Mastitis—postpartum				2		
Pleurisy diaphragmatic				1		
Section—uterine fibroids				1		
Diabetes mellitus				4		
Ureter—kink of				1		
Thrombophlebitis—legs				1		
Mole—hydatidiform				2		
Rheumatic—cardiovascular disease				4		
Pregnancy multiple				4		
Secundines retained				1		
Unregistered service					143	
White	77					
Colored	66					
Normal				57		

Abortion			7
Complete		7	
Toxemia			13
Eclampsia		4	
Preeclampsia		3	
Mild	1		
Severe	1		
Fulminating	1		
Hypertensive cardiovascular disease		4	
Mild	1		
Moderately severe	2		
Severe	1		
Hyperemesis gravidarum		2	
Multiparity			1
Labor false			4
Labor premature			7
Presentation transverse			1
Section previous			1
Cord prolapse of			1
Hemorrhage—vaginal			18
Antepartum		16	
Postpartum		2	
Placenta retained			1
Placenta adherent			2
Presentation breech			6
Membranes—premature rupture of			7
Endometritis—postpartum			2
Secundines retained			1
Pyelitis antepartum			3
Observation			3
Sclerosis multiple			2
Chorea—Huntington's severe			1
Obesity			1
Uterus—rupture of			1
Arm—prolapse of			1
Perineum—laceration of			1
Presentation compound—disproportion cephalopelvic			1

Number of patients transferred			13
Surgery		5	
Obstruction intestinal	1		
Mastitis	1		
Thrombophlebitis—leg—left	1		
Infection incisional	1		
Peritonitis—pneumococcus	1		
Medicine		5	
Hip—tuberculosis of	1		
Hepatitis—infectious—acute	1		
Tuberculosis pulmonary	1		
Septicemia—B coli	1		
Diabetes mellitus	1		

Gynecology			3	
Tuberculosis renal		1		
Fibroids—uterine		1		
Infection—uterine		1		
Number of patients admitted				2515
Number of patients delivered for other services				4
Surgery			1	
Obstruction—intestinal		1		
Medicine			3	
Arthritis gonorrheal		1		
Cord—spinal—transverse section of		1		
Tuberculosis		1		
Complications—inside and outside services				3887
Maternal			3746	
Placental		97		
Placenta previa centralis	6			
Placenta previa partialis	3			
Placenta previa marginalis	3			
Placenta abruptio	2			
Placenta—premature separation of—low implanted	13			
Placenta—premature separation of—normally implanted	31			
Placenta retained	14			
Placenta adherent	19			
Cord—prolapse of	6			
Abnormal presentation and abnormal pelvis		594		
Occiput posterior	160			
Occiput transverse	230			
Presentation breech	95			
Presentation transverse	9			
Presentation brow	3			
Presentation face	8			
Presentation compound	1			
Disproportion cephalopelvic	28			
Pelvis—contraction of	60			
Labor		664		
Premature	119			
Precipitate	54			
False	88			
Prolonged	193			
Shock intrapartum	6			
Shock postpartum	12			
Uterus—inertia of	2			
Ring—contraction—Bandl's	9			
Uterus—rupture of	2			
Uterus—inversion of	1			
Uterus—bicornute	1			
Dystocia cervical	15			
Membranes—rupture of—premature	162			

Hemorrhage and blood dyscrasia			94
Anemia secondary		52	
Hemorrhage antepartum		11	
Hemorrhage intrapartum		4	
Hemorrhage postpartum		27	
Cardiovascular disease			21
Rheumatic		21	
Mother—Rh negative			114
Toxemia			198
Hypertensive disease		87	
Eclampsia		7	
Antepartum	6		
Postpartum	1		
Preeclampsia		87	
Nephritis chronic		3	
Hyperemesis gravidarum		14	
Infection			415
Genital tract		284	
Infection puerperal	235		
Infection intrapartum	6		
Perineum—infection of	3		
Cervicitis	13		
Vaginitis and vulvitis	27		
Urinary tract		73	
Pyelitis antepartum	45		
Pyelitis postpartum	19		
Cystitis antepartum	6		
Cystitis postpartum	2		
Kidney—tuberculosis of	1		
Respiratory			30
Pneumonia—broncho		4	
Tuberculosis pulmonary		13	
Active	2		
Arrested	11		
Bronchitis acute		2	
Bronchiolitis acute		1	
Respiratory—acute—upper		1	
Tonsillitis acute		4	
Asthma—chronic		2	
Pneumonitis		3	
Miscellaneous infection			28
Mastitis—non-suppurative		12	
Mastitis suppurative		2	
Thrombophlebitis		4	
Abdominal wound—infection of		2	
Peritonitis—pelvic		1	
Disease—inflammatory—pelvic		2	
Arthritis—gonorrheal		1	
Tooth—abscess of		2	
Otitis media		1	
Fever—unknown origin		1	

Vaginal outlet—relaxed		66
Miscellaneous complications		1483
Vaginal mucous membrane laceration of	78	
Cervix—laceration of	165	
Perineum—laceration of	287	
Breasts—lesions of	1	
Legs—cramps of	13	
Measles	1	
Postmaturity	1	
Rectum—laceration of	54	
Pregnancy multiple	37	
Hydramnios	5	
Perineum—hematoma of	4	
Abortion missed	1	
Abortion incomplete	3	
Abortion threatened	21	
Fistula—rectovaginal	1	
Bleeding—vaginal—cause undetermined	28	
Cervix—erosion of	112	
Mole—hydatidiform	2	
Varicose veins—thrombosis of	4	
Psychosis postpartum	3	
Section previous	21	
Multiparity	35	
Secundines retained	6	
Hematuria—cause unknown	3	
Urine—retention of	3	
Glycosuria of pregnancy	1	
History—obstetric—poor	2	
Ureter—kink of	1	
Cervix—polyps of	1	
Ovary—dermoid cyst of	1	
Ovary—cysts of	2	
Vagina septum of	2	
Labor—arrested second stage	1	
Diabetes mellitus	6	
Paralysis agitans	2	
Sclerosis multiple	3	
Abdomen—mass of	1	
Uterus—fibroids of	6	
Atrophy muscular	1	
Polyneuritis	1	
Cervix—carcinoma of	1	
Malnutrition	1	
Pain—abdominal—obstruction intestinal	1	
Observation	14	
Cardiospasm	1	
Pleurisy diaphragmatic	1	
Chorea—Huntington's	1	
Obesity	2	

Epilepsy		4		
Condyloma acuminata		9		
Rectum—stricture of		1		
Deficiency mental		7		
Lordosis—lumbar		1		
Lower extremities—atrophy of post polio		1		
Lymphogranuloma venereum		2		
Anemia		439		
Schizophrenia		3		
Neuronitis		5		
Neurosis		1		
Pruritis		6		
Ani	2			
Vulvae	4			
Scabies		1		
Pediculosis		1		
Sinusitis		1		
Ribs—fracture of		1		
Imbecility		1		
Goitre		1		
Veins—varicose		22		
Duct—Skeen's—abscess of		1		
Cyst—Bartholin		11		
Cyst—Gartner's duct		2		
Hernia—umbilical		4		
Hemorrhoids		16		
Complications fetal				141
Injuries and diseases			49	
Hemorrhage intracranial		10		
Hemorrhage subarachnoid		3		
Asphyxia intrauterine		1		
Atelectasis		12		
Humerus—fracture of		1		
Parietal bone—fracture of		3		
Scalp—hematoma of		2		
Fever—dehydration and inanition		5		
Hypoglycemia		2		
Congenital hemolytic disease of newborn		9		
Burns—1st—2nd and 3rd degree of head—legs and arms		1		
Infection			78	
Diarrhea		17		
Thrush		13		
Impetigo		24		
Conjunctivitis		7		
Nonspecific	6			
Gonococcal	1			
Pneumonia		6		
Syphilis congenital		1		
Meningitis		1		
Scalp—cellulitis of		3		

Fever—origin unknown			1		
Infection—cord—umbilical			3		
Peritonitis			1		
Otitis media—bilateral			1		
Developmental				14	
Spina bifida with hydrocephalus			1		
Anencephalus			3		
Idiocy—Mongolian			1		
Talipes equino varus			2		
Anus—imperforate			1		
Heart—congenital			1		
Polydactylism			2		
Arm and forearm—left—absence of			1		
Anomalies—urinary			1		
Neck—triangle of—anterior—tumor malignant			1		
Number of viable babies					2722
In homes				658	
White	11				
Colored	647				
In hospital				2064	
White	1245				
Colored	819				
Full term living					2542—90.31%
In homes				626	
White	8				
Colored	618				
In hospital				1916	
White	1180				
Colored	736				
Full term dead					38—1.4%
In homes			4		
White	0				
Colored	4				
Cause undetermined		4			
In hospital			34		
White	11				
Colored	23				
Congenital hemolytic disease of newborn		2			
Placenta—premature separation of		3			
Disproportion—cephalo pelvic—outlet		2			
Cause undetermined		2			
Lues—maternal		3			
Dystocia—shoulder—post—maturity		2			
Infection—intrauterine antepartum		1			
Anencephalus		1			
Placenta abruptio		1			
Cord—prolapse of		4			
Presentation—breech—head aftercoming—arrest of		3			

Uterus—rupture of		1		
Arm—prolapse of		1		
Uterus—bicornute—presentation transverse		1		
Presentation face		1		
Hemorrhage intracranial		1		
Placenta previa—marginalis associated with hemorrhage		1		
Toxemia maternal		4		
Premature living				106—3.89%
In homes			19	
White	3			
Colored	16			
28 weeks spontaneous		2		
30 weeks spontaneous		5		
32 weeks spontaneous		3		
33 weeks spontaneous		2		
34 weeks spontaneous		2		
35 weeks spontaneous		1		
36 weeks spontaneous		3		
38 weeks spontaneous		1		
In hospital			87	
White	42			
Colored	45			
35 weeks spontaneous		4		
33 weeks presentation breech		1		
34 weeks preeclampsia		4		
34 weeks placenta premature separation of		2		
36 weeks spontaneous		14		
36 weeks preeclampsia		1		
36 weeks hypertensive cardiovascular disease		1		
34 weeks spontaneous		5		
32 weeks placenta previa centralis		2		
32 weeks preeclampsia		2		
30 weeks membranes premature rupture of		2		
30 weeks—pregnancy multiple		2		
37 weeks congenital hemolytic disease		1		
38 weeks spontaneous		3		
28 weeks spontaneous		3		
30 weeks spontaneous		4		
30 weeks placenta premature separation of		3		
32 weeks spontaneous		3		
34 weeks measles		1		
36 weeks placenta premature separation of		2		
36 weeks presentation breech		1		
28 weeks membranes premature rupture of		3		
36 weeks placenta abruptio		1		
36 weeks diabetes mellitus		1		
31 weeks diabetes mellitus		1		
38 weeks membranes premature rupture of		1		
32 weeks—bleeding vaginal cause undetermined		1		

28 weeks placenta premature separation of		1		
34 weeks congenital hemolytic disease		2		
36 weeks—cesarean section suspected disproportion		1		
32 weeks placenta premature separation of		1		
34 weeks spontaneous		2		
34 weeks hypertensive cardiovascular disease		1		
30 weeks placenta previa centralis		1		
38 weeks pregnancy multiple		4		
30 weeks lues maternal		1		
32 weeks pregnancy multiple		2		
32 weeks eclampsia		1		
30 weeks preeclampsia		1		
Premature dead				36—1.35%
In homes			9	
White	1			
30 weeks cause unknown		1		
Colored	8			
38 weeks cause unknown		1		
34 weeks cause unknown		1		
30 weeks lues maternal		1		
32 weeks lues maternal		1		
32 weeks cause unknown		1		
30 weeks cause unknown		3		
In hospital			27	
White	11			
30 weeks presentation transverse		1		
28 weeks cause unknown		1		
34 weeks preeclampsia		1		
36 weeks placenta—premature separation of		1		
30 weeks cause unknown		1		
32 weeks placenta premature separation of		2		
32 weeks preeclampsia		1		
32 weeks membranes premature rupture of		1		
32 weeks eclampsia		1		
34 weeks anencephalus		1		
Colored	16			
30 weeks placenta previa partialis		1		
32 weeks placenta premature separation of		2		
34 weeks labor prolonged 2nd stage		1		
28 weeks placenta premature separation of		2		
28 weeks lues maternal		1		
30 weeks cause unknown		1		
32 weeks presentation transverse—arm prolapse of		1		
32 weeks cause unknown		1		
34 weeks preeclampsia		1		
34 weeks placenta premature separation of		2		
33 weeks placenta premature separation of—second twin		1		
36 weeks eclampsia—post mortem section		1		
30 weeks eclampsia		1		

Deaths in live born babies				77—2.82%
In homes			4	
White	0			
Colored	4			
Lues maternal		1		
Prematurity		3		
In hospital			73	
White	33			
Prematurity		10		
Prematurity and eclampsia		1		
Prematurity and preeclampsia		1		
Tumor—neck—malignant		1		
Hemorrhage with associated asphyxia		1		
Hemorrhage subarachnoid		6		
Congenital hemolytic disease		4		
Diabetes mellitus—maternal and prematurity		1		
Skull—fracture of—hemorrhage subarachnoid		1		
Atelectasis		3		
Anomalies—congenital multiple		1		
Spina bifida and meningocele		1		
Pneumonia—broncho		1		
Diarrhea		1		
Colored	40			
Pneumonia—lobar—bilateral		1		
Hemorrhage intracranial		5		
Infection intrapartum		1		
Preeclampsia and prematurity		1		
Hemorrhage subarachnoid		1		
Lues congenital		2		
Pneumonia broncho		3		
Cause unknown		3		
Prematurity		9		
Congenital hemolytic disease		2		
Atelectasis		10		
Diarrhea		2		

Summary of causes of death in live born babies:	White	Colored	Total
Prematurity—main cause	13	13	26
Hemorrhage—intercranial	7	6	13
Atelectasis congenital	3	10	13
Congenital hemolytic disease	4	2	6
Infection acute	1	5	6
Lues	0	3	3
Anomalies congenital	3	0	3
Diarrhea	1	2	3
Asphyxia	1	0	1
Undetermined	0	3	3
	—	—	—
	33	44	77

Total fetal mortality			151—5.54%
In homes		17	
In hospital		134	
White	56—4.45%		
Colored	95—6.47%		

Maternal mortality			5—0.180%
In homes		0	
In hospital		5	
White	2—1.59 per 1000 live births		
Eclampsia		1	
Renal failure with associated peritonitis		1	
Colored	3—2.04 per 1000 live births		
Uterus—rupture of—pulmonary edema—acute		1	
Bronchiolitis acute—pulmonary edema—acute		1	
Eclampsia		1	
Deaths in registered clinic patients		2	
Deaths in unregistered clinic patients		3	
Deaths in private patients		0	
Rate per 1000 live births		1.88	

Number of S.T.S. in registered clinic patients			1792
White	291		
Colored	1501		

Number of positive results			223—12.44%
White	8—2.74%		
Colored	215—13.12%		

Number of positive results treated			216
White		9	
Colored		207	
Full term alive		194—90%	
Full term dead		5	
Undelivered		8	
38 weeks premature alive		1	
36 weeks premature alive		1	
34 weeks premature alive		2	
33 weeks premature alive		1	
32 weeks premature alive		1	
30 weeks premature alive		2	
38 weeks premature dead		1	

Number of positive results not treated			7
White	0		
Colored	7		
Full term alive		7—100%	

Number of patients without S.T.S.	0

SUMMARY OF MATERNAL DEATHS

91852 A 24 year old, colored, registered, para 5–0–0–5, admitted to the hospital for delivery of second twin. Delivered under sodium pentothal during which time there was a respiratory arrest of approximately five minutes. However she responded well to treatment, received 500 cc of blood during procedure. On her third postpartum day temperature suddenly rose to 102 with a pulse of 110 and respirations of 36. She was extremely dyspneic and brought up considerable sputum. Despite adequate treatment, including continuous oxygen, aminophyllin, plasma, penicillin and sodium iodide, course was rapidly downhill and respirations ceased on her 5th postpartum day 4 P.M., May 13, 1946. Tentative diagnosis: Bronchiolitis acute, Embolus pulmonary?

Cause of Death: Bronchiolitis acute.

Autopsy refused

94492 A 34 year old, colored, para 7–0–0–6, admitted to the hospital following unsuccessful attempts at delivery by her family physician on Aug. 6, 1946. Examination on admission revealed a fully dilated cervix, presenting part level with the spines and with a tense, firm uterus which relaxed between pains. Fetal heart not heard—position R.O.T. She complained bitterly of pain and while waiting to terminate the pregnancy, the picture was suddenly altered. Pain ceased, the presenting part ascended out of the pelvis and fetal parts could be easily palpated in the abdominal cavity. A laparotomy was performed, baby found free in the abdominal cavity with uterus well clamped down and lying in the right lower quadrant of the abdomen. Further examination revealed an extensive tear involving the left broad ligament, extending posteriorly into the cervix. A supravaginal hysterectomy was performed. She received 1000 cc of blood during the procedure. Postpartum course stormy and death occurred at 11.50 P.M. Aug. 9, 1946, 72 hours postoperative.

Cause of death: Uterus—rupture of.

Autopsy: Ventricle, left, acute dilatation of
Edema, pulmonary, acute
Uterus, rupture of.

97889 A 44 year old, white, para 12–0–0–8, admitted from the prenatal clinic because of hypertension and multiparity. Little improvement noted with routine treatment. Two medical inductions were unsuccessful. Labor began spontaneously on Dec. 7, 1946, little progress being made. Sent for flat plate of abdomen and the report revealed a transverse position, anterior posterior variety. Attempts made to convert same into breech by application of an abdominal binder without success. Labor was hard and unsuccessful and after 14 hours of such labor she was delivered of a living male child, weighing 8 lbs. 3½ oz. from L.S.A. position by classical cesarean section. Went into shock on her 5th postoperative day. Treated with oxygen, penicillin, blood and plasma. She developed anuria, went into coma and death occurred nine days following operation at 11.23 A.M. on Dec. 16, 1946.

Cause of death: Renal failure
Peritonitis.

Autopsy: Localized peritoneal abscess at operative site
Paralytic ileus
Pyelonephritis, moderate.

97150 A 25 year old white, unregistered, para 0–0–1–0, admitted to the hospital because of convulsions. Intensive therapy was instituted with little response following two convulsive seizures after admission. Blood chemistries gradually rose, anuria developed and she went into shock on Nov. 6, 1946 which was treated with plasma and routine therapy. She spontaneously went into labor and was delivered of a 32 weeks, dead male child, weighing 3 lbs. on Nov. 7, 1946. Postpartum course was rapidly downhill and death occurred at 2.26 P.M. Nov. 8, 1946, 24 hours following delivery.

Cause of death: Eclampsia, fulminating

Autopsy refused.

93770 A 30 year old, colored, unregistered, para 5–0–0–5, admitted to the hospital because of convulsions. Intensive therapy instituted with no response. Condition was rapidly downhill, blood pressure rose to 206, pulse 140, respirations 100, and at 8.19 A.M. on July 15, 1946 respirations ceased. A postmortem section was done but baby was stillborn.

Cause of death: Eclampsia.

Autopsy: Pale, swollen kidneys; cloudy swelling, liver marked; hydrothorax; anasarca; pulmonary congestion and edema.

MATERNAL MORTALITY COVERING NINE YEAR PERIOD

	1938	1939	1940	1941	1942	1943	1944	1945	1946
Peritonitis and uremia									1
Pregnancy, abdominal	1								
Abruptio placenta	2	1	1						
Shock, intrapartum (without hemorrhage)	1								
Embolus, pulmonary	1							1	
Infection, puerperal	1		2	1	1				
Uremia	1								
Reaction, transfusion	1								
Anesthetic	1	1	2			2		1	
Heart disease, rheumatic-decompensation	1		1				1	1	
Liver, acute yellow atrophy of		1							
Cardiovascular collapse		1	1	1	1	1			
Cardiac dilatation		1	1		1				
Uterus, rupture of		1	1	1		1		1	1
Hemorrhage, intracranial			1	2					
Arsphenamine encephalitis				1					
Tuberculosis				1	1		1		
Embolus, cerebral				1					
Leukemia, acute myeloid				1					
Eclampsia, ante and intrapartum				1			3		2
Hemorrhage, postpartum						1	1		
Ileus, paralytic					1				
Adrenal gland, insufficiency of					1				
Preeclampsia, severe, with shock							1		
Placenta previa								1	
Bronchiolitis, acute									1
Total 64	10	6	10	10	6	5	7	5	5
Rate per 1000 Live Births	5.4	3.0	4.4	3.9	2.0	1.6	2.4	2.19	1.89

POST GRADUATE ACTIVITIES OF THE SCHOOL OF MEDICINE, UNIVERSITY OF MARYLAND

April, 1947

HOWARD M. BUBERT, M.D.*†

Chairman, Post Graduate Committee, School of Medicine

More and more, progressive Medical Schools throughout the country are appreciating and accepting a responsibility in the field of Post Graduate Education. This is amply evidenced by a survey of eleven representative Medical Schools which revealed that the group in question was offering a total of eighty-three courses. Several contributing factors are operating to bring this about, namely: the increasingly high level of advanced education, generally; the demand from practicing physicians for instruction in the newer techniques, particularly those arising first, from the introduction of mechanical devices designed to improve diagnosis and therapy and, secondly, from the introduction of powerful antibiotic agents; and, the more stringent requirements that must be met by physicians desiring certification in any one of the various specialties.

This Committee being, as it is, in its infancy must limit its activities to those phases of post graduate education coming within the scope of its authority and resources. In this discussion, we are concerned with the need existing for extra mural teaching of practicing physicians working in areas remote from the usual intra mural educational facilities. Immediately, several questions present themselves for consideration.

The matter of expense is of major importance, in our case, at least. No provision has been made in the budget of this Medical School for these activities, consequently, some fee would of necessity have to be charged to cover, at least partially, the cost of the project. However, it is recognized that it must be moderate so that the largest possible number might avail themselves of its possibilities.

The type course is also a factor of consequence. It seems to us that one "work-day" might be allotted to self improvement by even the busiest physician, if he sincerely desired to participate. This day's instruction could be repeated at varying intervals until the subjects to be considered are adequately covered.

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* The foregoing represents the official viewpoint of the Post Graduate Committee upon the subject under discussion.

† Comment and suggestions are urgently solicited. They should be addressed to the Post Graduate Committee, Attention, Mrs. Elizabeth Carroll, Secretary to the Committee, 29 S. Greene Street, Baltimore 1, Maryland.

The content of the course is, also, of prime interest. Should such a project prove feasible, we believe that the local group of potential participants might well express their wishes in this regard. Whereas, it might not be practicable to follow exactly their proposals, certainly they would serve as a useful guide to the Committee in arranging a popular and useful schedule.

The Committee recognizes the burden of work placed upon the Medical School teaching staff. However, it feels that the obligation resting upon a State Medical School to supply this type service, is so heavy that necessary adjustments must be made in the staff to enable the school to discharge it.

If the challenge is not accepted, we believe that we may rest assured that the demand will not become less. On the contrary, we believe that either we will be forced to supply the service upon terms dictated by others, or that another agency will be given the staff, the facilities and the responsibility for the completion of the task.

The Committee is most anxious to receive expressions of opinion from interested physicians, particularly those likely to be interested in securing instruction of this type, as such expressions of opinion will be of immeasurable assistance to the Committee in its attempt to explore this field intelligently.

TO ALL GRADUATES OF THE MEDICAL SCHOOL

In 1813 the Medical Faculty of the University of Maryland bought the books of Dr. John Crawford to start a medical library, one of the oldest in the country. This book collection, remarkable at the time, cost five hundred dollars; today, even more remarkable because of the age and rarity of many volumes, it is worth thousands.

Unfortunately, these valuable books were so neglected over a long period of years that the bindings fell into critical disrepair. The same neglect affected, also, a large number of volumes in the library's general historical collection of sixteenth, seventeeth, and eighteenth century medical books.

To restore the bindings and thus preserve these books, the library needs extensive funds not available in the regular budget. If it is a luxury to save a significant collection of rare books and to restore them to creditable condition, then the library feels justified in asking for luxury funds. Such funds would be set up in a restricted account, to be used only at the direction of the Library Committee for the express purpose stated.

To you we appeal for an annual contribution for five years, in any amount; or if you prefer, a single contribution. Yearly notice will be sent to those pledging contributions. A roster of contributors' names will be made for display in the historical room of the library and for publication in the Bulletin of the School of Medicine, to show our alumni's tangible interest in historical medicine.

John E. Savage, M.D., Chairman
Book Restoration Committee

Date........................

I pledge an annual contribution of $—— for five years for the restoration of the rare books of the Medical Library. Enclosed is my 1947 contribution.

Name: ....
Address: ....
....

Please make checks payable to John E. Savage, Chairman, and mail to the Medical Library, University of Maryland, S. E. Corner Lombard & Greene Streets, Baltimore 1.

MONTHLY MEETINGS OF THE UNIVERSITY HOSPITAL STAFF

The following is an abstract of a paper presented at the monthly meeting of the University Hospital staff held on January 16, 1947.

CARCINOMA OF THE TONGUE

A. J. Siwinski, M.D.*

A statistical study was presented of carcinoma of the tongue treated at the University Hospital clinic and the Johns Hopkins clinic and in the private practise of Dr. Grant E. Ward. The work was divided into two phases, those cases treated from 1925 to 1940 and those from 1940 to 1945.

1925 to 1940	Total	Alive & well	Dead	Not followed
Surgery alone	14	1	12	1
Irradiation less than 10 TED (Tissue erythema dose)	48	3	35	10
Irradiation more than 10 TED	3	1	1	1
Surgery and irradiation more than 10 TED	1	1	0	0
1940 to 1945				
Surgery alone	13	5	8	0
Irradiation less than 10 TED	17	0	14	3
Irradiation more than 10 TED	5	4	1	0
Surgery and irradiation more than 10 TED	8	6	2	0

It will be noticed the greatest mortality was in cases treated with surgery alone and inadequate irradiation. Adequate dosage is considered as 10 TED which means that this dose is delivered to the growth itself. The second group was added not because it was felt that sufficient time has elapsed since their treatment, but because in the group treated by irradiation and surgery it was found (on serial pathologic section) that the tumor had been completely erradicated with adequate irradiation. For these reasons the following course is advocated for all carcinoma of the tongue.

1. Adequate irradiation;
    a. Roentgen ray-intra oral and external
    b. Radon seeds alone and in combination with a.
2. Hemiglossectomy
3. Radical neck dissection whether nodes are palpable or not.

Dr. Kilby then discussed the paper advocating close cooperation of the surgeon with the radiologist.

The following is an abstract of a paper presented at the monthly meeting of the University Hospital staff held on April 17, 1947.

TOXOPLASMOSIS—A SUMMARY

F. Edwin Knowles, Jr., M.D.†

Toxoplasmosis is a recently recognized infectious disease in human beings first proven by demonstration of toxoplasmas at autopsy in 1939 in the central nervous system and the eye in an infant with encephalomyelitis. The mode of transmission from animals to man is unknown although animal toxoplasmosis is considered to be the source of the human disease.

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* Department of Surgery, School of Medicine, University of Maryland.

† Department of Ophthalmology, School of Medicine, University of Maryland.

The principal clinical findings are:

1. Acute encephalomyelitis in infants and children with internal hydrocephalus or microcephaly
2. Roentgenographic evidence of focal cerebral calcification
3. Bilateral chorioretinitis

Ocular involvement is an early and prominent feature and this, associated with clinical signs and general symptoms of toxoplasmosis, help to establish the diagnosis of this condition.

The following are abstracts of papers presented at the monthly meeting of the University Hospital staff held on May 15, 1947.

DISCUSSION OF SOME OF THE NEW FUNGICIDES

Eugene S. Bereston, M.D.*

Arenson, while working with the problem of food preservation, discovered that the esters of the higher unsaturated fatty acids were fungicidal to most of the common fungi which were pathogenic to man. His in vitro studies showed propylene glycol di proprionate was the least toxic and the best fungicide. This compound on repeated clinical tests was found to be a good fungicide in a variety of cutaneous conditions, but was not successful in Tinea Capitis.

Propylene glycol di pelargonate was used in Tinea Capitis with slightly better results than the di proprionate, but neither compound is satisfactory in Tinea Capitis.

ELICITATION OF CONTACT INFORMATION IN VENEREAL DISEASE CONTROL

I. ZELIGMAN, M.D.*

Venereal Disease Control is a very important aspect of preventive medicine. There are many necessary factors in the prevention of venereal diseases such as: Proper diagnosis and treatment, educational measures, prophylaxis, improved economic and social status and contact information. Because the venereal diseases are spread almost exclusively by sexual intercourse it is essential to trace and treat the sexual contacts in order to prevent additional spread of the infection to others. The proper method of obtaining this information consists of a personal confidential chat with the patient, during which talk the patient is taught the facts of his disease and methods of preventing the disease in himself and others. Finally contact information is elicited when the patient fully understands its importance to himself and the community.

IN VITRO STUDIES OF MICROSPORON AUDOUINI INFECTION OF THE HAIR

Harry M. Robinson, Jr., M.D.*

Experiments were performed, testing the effect of various so-called fungicides on Microsporon Audouini infection of the hair. The results indicate that the term fungistatic should probably be used, rather than fungicide.

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* Department of Dermatology, School of Medicine, University of Maryland.

PROCEEDINGS

of the
University of Maryland Biological Society

The following is an abstract of a paper presented at a meeting of the University of Maryland Biological Society on April 16, 1947.

THE ANESTHETIC PROPERTIES OF ALIPHATIC ETHERS

C. Jelleff Carr, Ph.D. and John C. Krantz, Jr., Ph.D.*

A statistical study of the anesthetic potency and safety of elevent ethers has been carried out on the dog.

The data for the margin of safety of most of the ethers cluster themselves closely about the arbitrary standard, the arithmetic mean ethyl ether.

Striking exceptions are isopropyl methyl which has a wide margin of safety, and divinyl oxide whose margin of safety appears inordinately low.

The place of the halogenated hydrocarbon chloroform in this series is discussed.

NEWS ITEMS

Captain Benjamin Amsterdam, Medical Corps, United States Army, was recently promoted to the rank of captain at Tilton General Hospital, Fort Dix, New Jersey, where he is consulting medical officer with the Internal Medicine Section of Medical Service.

The annual Phi Delta Epsilon Lecture, sponsored by the Delta Epsilon Chapter at the University of Maryland School of Medicine, was given by Dr. Priscilla White of Boston, on the subject "Diabetes and Pregnancy." This lecture was given on March 24th at 8:00 P.M. in Chemical Hall to an overflowing crowd of Seniors and Juniors of the School of Medicine and to physicians of the city.

Dr. Priscilla White is a graduate of Tuft's Medical College and is a member of the faculty there. Since 1923, she has been associated with Dr. Joslin's Clinic in Boston, and has devoted her special interest to diabetes in childhood, adolescence, and pregnancy. She is one of the collaborators with Dr. Elliott Joslin in the publishing of Dr. Joslin's book on the "Treatment of Diabetes."

Preceding the lecture a dinner was given in honor of Dr. Priscilla White, at the Southern Hotel. Among those who attended were Dr. Maurice Pincoffs, Dr. L. A. Krause, Dr. Louis H. Douglass, Dr. J. E. Gichner, Dr. N. J. Eastman, Dr. A. E. Goldstein, Dr. S. S. Glick, Dr. I. A. Siegel, Dr. Alan F. Guttmacher, and others.

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* Department of Pharmacology, School of Medicine, University of Maryland.

Dr. I. A. Siegel, Assistant Professor of Obstetrics at the University of Maryland, was the chairman for the Phi Delta Epsilon Lecture and presided at the meeting.

Dr. Louis H. Douglass, Professor of Obstetrics, introduced the speaker.

ERRATUM

In the Index to Volume 31 of the Bulletin it was listed that on page 41 there was an obituary of Ruth Lee Briscoe. The article on Miss Briscoe was a tribute, not an obituary. The Bulletin regrets the error.

OBITUARIES

Anderson, John, Jr., New York, N. Y.; P. & S., class of 1913; aged 58; died, January 19, 1947, of hemoperitoneum following an automobile accident.

Bogar, Joseph E., Rahway, N. J.; P. & S., class of 1892; aged 75; died, January 21, 1947, of cerebral hemorrhage.

Chapman, George Andrew, Glens Falls, N. Y.; B.M.C., class of 1897; aged 75; died, December 16, 1946, of uremia.

Clark, Bruce, Hiwassee, Va.; P. & S., class of 1891; aged 80; died, December 19, 1946, of tuberculosis.

de la Vega, Luis B., San Juan, P. R.; P. & S., class of 1914; aged 58; died, February 9, 1947, of cerebral hemorrhage.

Duker, Otto H., Baltimore, Md.; P. & S., class of 1904; aged 79; died, January 19, 1947, of arteriosclerosis and thrombosis of femoral artery.

Dye, Frank Ganes, Skaneateles, N. Y.; class of 1919; aged 54; served during World War II; died, January 5, 1947, of cerebral hemorrhage.

Fancher, H. Wilson, Thompsonville, Conn.; class of 1925; died, March 19, 1947, of coronary thrombosis.

Gibson, John Aldridge, Leesburg, Va.; class of 1901; aged 70; died, March 10, 1947, of carcinoma of the gall bladder.

Grizzard, James Alexander, Drewryville, Va.; B.M.C., class of 1906; aged 65; died, January 28, 1947, of lymphatic leukemia.

Hartman, James Henry, Jacksonville, Fla.; P. & S., class of 1904; aged 66; died, December 26, 1946, of coronary occlusion.

Hawkins, Vallandingham, Fawn Grove, Pa.; B.M.C., class of 1894; aged 82; died, December 13, 1946, of carcinoma.

Jarrell, Kyle McCue, Beckley, W. Va.; class of 1906; aged 65; died, January 24, 1947, of cerebral hemorrhage.

Koon, Thomas W., Cumberland, Md.; P. & S., class of 1893; aged 76; died, December 5, 1947.

McFarland, William Smith, Zanesville, O., P. & S., class of 1886; aged 83; died recently.

McReynolds, Alva Edgar, El Paso, Ill.; class of 1915; aged 70; served during World War I; died, February 6, 1947, of coronary occlusion.

Miller, Wilfred Porter, Syracuse, N. Y.; class of 1917; aged 61; died, January 16, 1947, of coronary thrombosis.

Morgan, Heber Guy, Red Lodge, Mont.; P. & S., class of 1906; aged 63; died, November 26, 1946, of carcinoma of the liver.

Myers, Benjamin Franklin, Chambersburg, Pa.; B.M.C., class of 1892; aged 78; died, February 17, 1947, of carcinoma of the stomach.

O'Mara, John T., Baltimore, Md. class of 1903; aged 66; died, February 3, 1947, of cerebral hemorrhage.

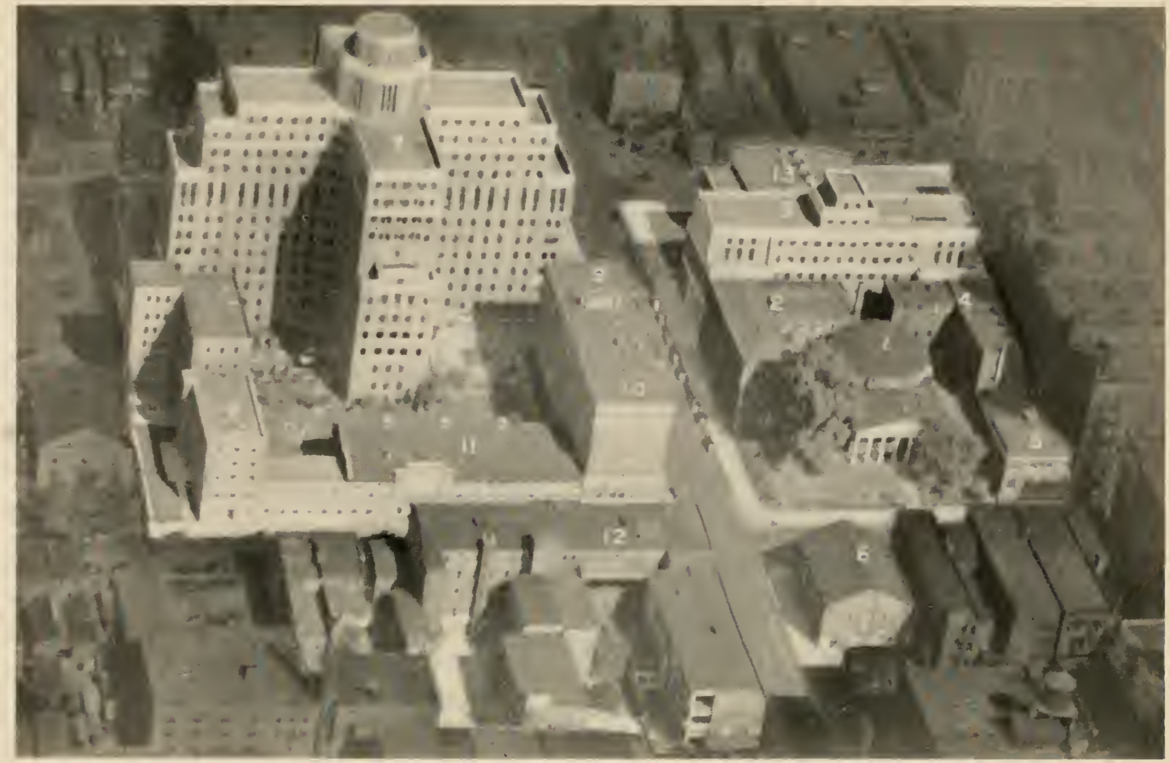
Patrick, Lytle Neale, Gastonia, N. C.; class of 1909; died, February 12, 1947, of a cerebral hemorrhage.

Reynolds, Knight, Cumberland, Md.; class of 1925; aged 47; died, February 5, 1947.

Saye, Joseph H., Sharon, S. C.; P. & S., class of 1885; aged 84; died, December 27, 1941, of heart disease.

Sprague, Fred Alvah, Concord, N. H.; B.M.C., class of 1906; aged 73; veteran of the Spanish American War; served during World War I; died, December 3, 1946, of cerebral hemorrhage.

Williams, Joel Govan, Norway, S. C.; B.M.C., class of 1890; aged 85; died, December 12, 1946, of heart disease.



BALTIMORE SCHOOLS—UNIVERSITY OF MARYLAND

1. Original Medical Building
2. Laboratory Building, Medicine
3. Bressler Building, Medicine
4. Gray Laboratory, Student's Lounge, Medicine
5. Administration Building, College of Education, Baltimore Division
6. Medical Library
7. University Hospital
8. Nurses' Home, Medicine
9. School of Pharmacy
10. School of Dentistry
11. Dental Clinic
12. Out-Patient Clinics, Medicine
13. School of Law

BULLETIN
OF THE
SCHOOL *of* MEDICINE
UNIVERSITY OF MARYLAND

SEPTEMBER, 1947

1807-1856-1920

Announcements for

The One Hundred Forty-Second Academic Session
1947–1948

Catalogue of
The One Hundred Forty-first Academic Session
1946–1947

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CALENDAR

1947

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CALENDAR

Academic Year—September 18, 1947 to June 5, 1948

1947		
September 10, 11, 12		Re-examinations for advancement

FIRST SEMESTER—September 18, 1947 to January 24, 1948

September 16	Tuesday	*Registration, payment of fees, freshmen and sophomores
September 17	Wednesday	*Registration and payment of fees by all other students
September 18	Thursday	Instruction begins at 8:30 A.M.
November 26	Wednesday	Instruction suspended at 5:00 P.M.
		Thanksgiving Holidays
December 1	Monday	Instruction resumed
December 20	Saturday	Instruction suspended at 2:00 P.M.
		Christmas Holidays
1948		
January 5	Monday	Instruction resumed
January 19	Monday	Midyear examinations, all classes
to		*Payment of fees for second semester
January 24	Saturday	First semester completed 2:00 P.M.

SECOND SEMESTER—January 26 to June 5, 1948

January 26	Monday	Instruction begins at 8:30 A.M.
February 21	Saturday	Instruction suspended at 2:00 P.M.
February 23	Monday	Holiday—Washington's Birthday
February 24	Tuesday	Instruction resumed
March 24	Wednesday	Instruction suspended at 5:00 P.M.
		Easter Holidays
April 1	Thursday	Instruction resumed
May 10	Monday	Senior examinations begin
May 17	Monday	Junior examinations begin
May 24	Monday	Junior examinations continue
		Sophomore and Freshmen examinations begin
May 29	Saturday	Announcement of graduates
June 5	Saturday	Commencement at 11:00 A.M.
		Second semester completed at 12:30 P.M.

PARTIAL CALENDAR FOR 1948–1949

September 8, 9, 10		Re-examinations for advancement
September 14	Tuesday	*Registration and payment of fees by freshmen and sophomores
September 15	Wednesday	*Registration and payment of fees by all other students
September 16	Thursday	Instruction begins at 8:30 A.M.

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* A student who fails to register prior to or within the day or days specified will be called upon to pay a late registration fee of five dollars ($5.00). The last day of registration with fee added to regular charges is Saturday of the week in which registration begins.

The offices of the registrar and comptroller are open daily from 9:00 A.M. to 5:00 P.M., and Saturday from 9:00 A.M. to 12:00 noon.

ORGANIZATION

THE UNIVERSITY OF MARYLAND

Harry Clifton Byrd, B.S., LL.D., D.Sc., *President and Executive Officer*

BOARD OF REGENTS

		Term Expires
William P. Cole, Jr., *Chairman*	Baltimore	1949
Thomas R. Brookes, *Vice-Chairman*	Bel Air	1952
Stanford Z. Rothschild, *Secretary*	Baltimore	1952
J. Milton Patterson, *Treasurer*	Baltimore	1953
E. Paul Knotts	Denton	1954
Glenn L. Martin	Baltimore	1951
Charles P. McCormick	Baltimore	1948
Harry H. Nuttle	Denton	1950
Philip C. Turner	Baltimore	1950
Millard E. Tydings	Washington, D. C.	1951
Mrs. John L. Whitehurst	Baltimore	1947

Members of the Board are appointed by the Governor of the State for terms of nine years each, beginning the first Monday in June.

The President of the University of Maryland is, by law, Executive Officer of the Board.

A regular meeting of the Board is held the third Friday in each month, except during the months of July and August.

Each school has its own Faculty Council, composed of the Dean and members of its faculty; each Faculty Council controls the internal affairs of the group it represents.

The University has the following educational organizations:

At Baltimore

- The School of Dentistry
- The School of Law
- The School of Medicine
- The School of Nursing
- The School of Pharmacy
- The College of Education (Baltimore Division)

At College Park

- The College of Agricultural
- The College of Arts and Sciences
- The College of Business and Public Administration
- The College of Commerce
- The College of Education
- The College of Engineering
- The College of Home Economics
- The Graduate School
- The Department of Military Science and Tactics
- The Department of Physical Education and Recreation
- The Summer School

FACULTY OF MEDICINE

EMERITI

J. Frank Crouch, M.D.....Professor of Clinical Ophthalmology and Otology, Emeritus
Harry Friedenwald, A.B., M.D., D.H.L., D.Sc..Professor of Ophthalmology, Emeritus
William S. Gardner, M.D. ....Professor of Gynecology, Emeritus
J. M. H. Rowland, M.D., D.Sc., LL.D.
Professor of Obstetrics, Emeritus; Dean, Emeritus
J. Dawson Reeder, M.D............................Professor of Proctology, Emeritus
Henry J. Walton, M.D.......................Professor of Roentgenology, Emeritus
Page Edmunds, M.D.....................Professor of Traumatic Surgery, Emeritus
Ruth Lee Briscoe..Librarian, Emeritus
Albertus Cotton, M.A., M.D...Professor of Orthopaedic Surgery, and Roentgenology, Emeritus
William Tarun, M.D.....................Professor of Ophthalmology, Emeritus
Joseph E. Gichner, M.D...... Professor of Clinical Medicine and Physical Therapeutics, Emeritus
Harvey G. Beck, M.D., D.Sc...............Professor of Clinical Medicine, Emeritus
Irving J. Spear, M.D.........................Professor of Neurology, Emeritus

FACULTY BOARD

H. Boyd Wylie, *Acting Dean*

William R. Amberson
Franklin B. Anderson
Rubert S. Anderson
James G. Arnold, Jr.
Thomas B. Aycock
Walter A. Baetjer
Charles Bagley, Jr.
J. McFarland Bergland
Charles F. Blake
Otto C. Brantigan
Howard M. Bubert
T. Nelson Carey
C. Jelleff Carr
Thomas R. Chambers
Carl Dame Clarke
Ross McC. Chapman
Clyde A. Clapp
Paul W. Clough
Richard G. Coblentz
Beverly C. Compton
Charles N. Davidson
Carl L. Davis
Louis H. Douglass
Frederick C. Dye
C. Reid Edwards
Monte Edwards
Frank H. J. Figge
Leon Freedom
Edgar B. Friedenwald
Thomas K. Galvin
Moses Gellman
Andrew C. Gillis
Henry F. Graff
Frank W. Hachtel
O. G. Harne
Harold E. Harrison
Cyrus F. Horine
J. Mason Hundley, Jr.
Elliott H. Hutchins
Edward S. Johnson
F. L. Jennings
C. Loring Joslin
Walter L. Kilby
Edward A. Kitlowski
Vernon E. Krall
John C. Krantz, Jr.
Louis A. M. Krause
Kenneth D. Legge
R. W. Locher
G. Carroll Lockard
Edward A. Looper
William S. Love, Jr.
John F. Lutz
Stanley H. Macht
Howard J. Maldeis
N. Clyde Marvel
Charles W. Maxson
James C. McAlpine
Zachariah Morgan
Theodore H. Morrison
Alfred T. Nelson
Emil Novak
Thomas R. O'Rourk
Robert H. Oster
C. W. Peake
D. J. Pessagno
H. R. Peters
Maurice C. Pincoffs
J. G. M. Reese
Charles A. Reifschneider
Dexter L. Reimann
Compton Riely
Harry M. Robinson, Sr.
Harry L. Rogers

Milton S. Sacks
Emil G. Schmidt
Arthur M. Shipley
Dietrich C. Smith
R. Dale Smith
William H. Smith
Hugh R. Spencer
Thomas P. Sprunt
W. Houston Toulson
Ralph P. Truitt
Eduard Uhlenhuth
Henry F. Ullrich
Allen F. Voshell
John A. Wagner
Grant E. Ward
C. Gardner Warner
Huntington Williams
Walter D. Wise
Thomas C. Wolff
Robert B. Wright
George H. Yeager
Waitman F. Zinn

EXECUTIVE COMMITTEE OF THE FACULTY

H. Boyd Wylie, *Acting Dean*

Louis H. Douglas
Charles Reid Edwards
Frank W. Hachtel
J. Mason Hundley, Jr.
Maurice C. Pincoffs
Arthur M. Shipley
Hugh R. Spencer

FACULTY OF MEDICINE

PROFESSORS

William R. Amberson, Ph.D., Professor of Physiology.[6]
Thomas B. Aycock, B.S., M.D., Professor of Clinical Surgery.
Charles Bagley, Jr., M.A., M.D., Professor of Neurological Surgery.
Charles F. Blake, M.A., M.D., Professor of Proctology.
Otto C. Brantigan, B.S., M.D., Professor of Surgical Anatomy, Associate Professor of Surgery.
T. Nelson Carey, M.D., Professor of Clinical Medicine[2] and Physician in Charge of Medical Care of Students.
Ross McC. Chapman, M.D., Professor of Psychiatry.
Clyde A. Clapp, M.D., Professor of Ophthalmology.
Richard G. Coblentz, M.A., M.D., Clinical Professor of Neurological Surgery.
Carl L. Davis, M.D., Professor of Anatomy.
Brice M. Dorsey, D.D.S., Professor of Oral Surgery, School of Dentistry.
Louis H. Douglass, M.D., Professor of Obstetrics.

It is to be noted that for convenience of reference the names of the members of the Faculty are listed in the forepart of this catalogue in alphabetical order. The names are listed in order of seniority under each preclinical and clinical department of the school on subsequent pages.

On the lists of the Faculty of Medicine and Fellows and the Hospital and Dispensary staffs are given the names and positions assigned during the period July 1, 1946 to June 30, 1947 unless otherwise indicated. Changes are noted as follows:

[1] Appointments effective July 1, 1947.
[2] Promotions effective July 1, 1947.
[3] Resignations effective July 1, 1947.
[4] On leave, effective July 1, 1947.
[5] Deaths.
[6] On leave, Sabbatical, May 16–November 15, 1947.

Charles Reid Edwards, M.D., Professor of Surgery.
Monte Edwards, M.D., Clinical Professor of Surgery and Proctology.
Frank H. J. Figge, Ph.D., Professor of Experimental Anatomy.[2]
H. K. Fleck, M.D., Clinical Professor of Ophthalmology.
Edgar B. Friedenwald, M.D., Professor of Clinical Pediatrics.
Thomas K. Galvin, M.D., Clinical Professor of Gynecology.
Andrew C. Gillis, M.A., M.D., LL.D., Professor of Neurology.
Frank W. Hachtel, M.D., Professor of Bacteriology.
J. Mason Hundley, Jr., M.A., M.D., Professor of Gynecology.
Elliott H. Hutchins, M.A., M.D., Professor of Surgery.
F. L. Jennings, M.D., Professor of Clinical Surgery.
C. Loring Joslin, M.D., Professor of Pediatrics.
Walter L. Kilby, M.D., Professor of Roentgenology.
Edward A. Kitlowski, A.B., M.D., Clinical Professor of Plastic Surgery.
John C. Krantz, Jr., Ph.D., D.Sc., Professor of Pharmacology.
Louis A. M. Krause, M.D., Professor of Clinical Medicine.[2]
Kenneth D. Legge, M.D., Professor of Clinical Genito-Urinary Surgery.
G. Carroll Lockard, M.D., Professor of Clinical Medicine.
Edward A. Looper, M.D., D.Oph., Professor of Rhinology and Laryngology.
Theodore H. Morrison, M.D., Clinical Professor of Gastro-Enterology.
Thomas R. O'Rourk, M.D., Clinical Professor of Otology, Associate Professor of Rhinology and Laryngology.[2]
Robert H. Oster, Ph.D., Professor of Physiology, School of Dentistry.
D. J. Pessagno, A.B., M.D., Professor of Clinical Surgery.
H. Raymond Peters, A.B., M.D., Professor of Clinical Medicine.
Maurice C. Pincoffs, B.S., M.D., Professor of Medicine.
Charles A. Reifschneider, M.D., Clinical Professor of Traumatic Surgery.
Compton Riely, M.D., Clinical Professor of Orthopaedic Surgery.
Harry L. Rogers, M.D., Clinical Professor of Orthopaedic Surgery.[2]
Harry M. Robinson, Sr., M.D., Professor of Dermatology.
Emil G. Schmidt, Ph.D., LL.B., Professor of Biological Chemistry.
Arthur M. Shipley, M.D., D.Sc., Professor of Surgery.
Hugh R. Spencer, M.D., Professor of Pathology.
Thomas P. Sprunt, A.B., M.D., Professor of Clinical Medicine.
W. Houston Toulson, M.Sc., M.D., Professor of Genito-Urinary Surgery.
Ralph P. Truitt, M.D., Professor of Clinical Psychiatry.[6]
Eduard Uhlenhuth, Ph.D., Professor of Anatomy.
Allen Fiske Voshell, A.B., M.D., Professor of Orthopaedic Surgery.
Huntington Williams, M.D., Dr. P.H., Professor of Hygiene and Public Health.
Walter D. Wise, M.D., Professor of Surgery.
H. Boyd Wylie, M.D., Professor of Biological Chemistry and Acting Dean.
Waitman F. Zinn, M.D., Clinical Professor of Rhinology and Laryngology.

ASSOCIATE PROFESSORS

Franklin B. Anderson, M.D., Associate Professor of Rhinology and Laryngology, and Otology.
James G. Arnold, Jr., M.D., Associate Professor of Neurological Surgery.
Walter A. Baetjer, A.B., M.D., Associate Professor of Medicine.

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[6] Promoted September 10, 1946.

J. McFarland Bergland, B.S., M.D., Associate Professor of Obstetrics.
J. Edmund Bradley, M.D., Associate Clinical Professor of Pediatrics.[2,4]
H. M. Bubert, M.D., Associate Professor of Medicine.[2]
C. Jelleff Carr, Ph.D., Associate Professor of Pharmacology.
Thomas R. Chambers, A.B., M.D., Associate Professor of Surgery.
Carl Dame Clarke, M.A., Associate Professor of Art as Applied to Medicine.
Paul W. Clough, B.S., M.D., Associate Professor of Medicine.
Charles N. Davidson, M.D., Associate Professor of Roentgenology.
Ross Davies, M.D., Associate Professor of Hygiene and Public Health.[2]
Frederick C. Dye, M.D., Associate Professor of Anaesthesiology.
Edward C. Dobbs, D.D.S., Professor of Pharmacology, School of Dentistry.
J. S. Eastland, M.D., Associate Professor of Medicine.[2]
A. H. Finkelstein, M.D., Associate Clinical Professor of Pediatrics.[2]
Leon Freedom, M.D., Associate Professor of Neurology.
Moses Gellman, B.S., M.D., Associate Professor of Orthopaedic Surgery.
T. Campbell Goodwin, M.S., M.D., Associate Professor of Pediatrics.[3]
Lewis P. Gundry, M.D., Associate Professor of Medicine.[2]
O. G. Harne, Associate Professor of Histology.
Harold E. Harrison, B.S., M.D., Associate Professor of Pediatrics.[1]
Horace Hodes, M.D., Associate Professor of Hygiene and Public Health.[1]
Cyrus F. Horine, M.D., Associate Professor of Surgery.
Harry C. Hull, M.D., Associate Professor of Surgery.[2]
Albert Jaffe, M.D., Associate Clinical Professor of Pediatrics.[2]
Edward S. Johnson, M.D., Associate Professor of Surgery.
Hugh W. Josephs, M.D., Associate Professor of Pediatrics.[3]
Vernon D. Kaufman, D.D.S., Associate Professor of Oral Surgery, School of Dentistry.
Vernon E. Krall, B.S., M.S., Associate Professor of Gross Anatomy.[6]
R. W. Locher, M.D., Associate Professor of Clinical Surgery.
William S. Love, Jr., A.B., M.D., Associate Professor of Medicine.
Howard J. Maldeis, M.D., Associate Professor of Legal Medicine and Associate in Pathology.
N. Clyde Marvel, A.B., M.D., Associate Professor of Surgery.
Charles W. Maxson, M.D., Associate Professor of Surgery.
James G. McAlpine, Ph.D., Associate Professor of Bacteriology.
Walter C. Merkel, A.B., M.D., Associate Professor of Pathology.[2]
H. W. Newell, M.D., Associate Professor of Psychiatry.[7]
Emil Novak, A.B., M.D., D.Sc., Associate Professor of Obstetrics.
C. W. Peake, M.D., Associate Professor of Surgery.
J. G. M. Reese, M.D., Associate Professor of Obstetrics.
Benjamin S. Rich, A.B., M.D., Associate Professor of Rhinology and Laryngology,[2] Associate in Otology.
Milton S. Sacks, M.D., Associate Professor of Medicine[2] and Head of Clinical Pathology, Associate in Pathology[1]
Dietrich Conrad Smith, Ph.D., Associate Professor of Physiology.[8]
Frederick B. Smith, M.D., Associate Clinical Professor of Pediatrics.[2]

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[4] On leave May 1, 1947 to April 30, 1948.
[6] Appointed August 1, 1947.
[7] Promoted September 10, 1946.
[8] On leave, Sabbatical, March 1–August 31, 1947.

William H. Smith, M.D., Associate Professor of Clinical Medicine.
Henry F. Ullrich, M.D., D.Sc., Associate Professor of Orthopaedic Surgery.
Grant E. Ward, A.B., M.D., Associate Professor of Surgery and Oral Surgery.
C. Gardner Warner, A.B., M.D., Associate Professor of Pathology.
William H. F. Warthen, A.B., M.D., Associate Professor of Hygiene & Public Health.[2]
J. Herbert Wilkerson, M.D., Associate Professor of Oral Surgery, School of Dentistry.
Thomas C. Wolff, M.D., Associate Professor of Medicine.[2]
Robert B. Wright, B.S., M.D., Associate Professor of Pathology.
George H. Yeager, B.S., M.D., Associate Professor of Surgery.

ASSISTANT PROFESSORS

Thurston R. Adams, M.D., Assistant Professor of Surgery and Proctology.
Rubert S. Anderson, Ph.D., Assistant Professor of Physiology.
H. F. Bongardt, M.D., Assistant Professor of Surgery.
Leo Brady, A.B., M.D., Assistant Professor of Gynecology.
Simon H. Brager, M.D., Assistant Professor of Surgery and Associate in Proctology.
Edward F. Cotter, M.D., Assistant Professor of Medicine,[1] Instructor in Neurology.[3]
Francis A. Ellis, A.B., M.D., Assistant Professor of Dermatology.
Maurice Feldman, M.D., Assistant Professor of Gastro-Enterology.
Wetherbee Fort, M.D., Assistant Professor of Medicine.[2]
Frank J. Geraghty, M.D., Assistant Professor of Medicine.[2]
Francis W. Gillis, M.D., Assistant Professor of Genito-Urinary Surgery.
Samuel S. Glick, M.D., Assistant Professor of Pediatrics.[2]
Harry Goldsmith, M.D., Assistant Professor of Psychiatry.[3]
Albert E. Goldstein, M.D., Assistant Professor of Pathology.
Henry F. Graff, A.B., M.D., Assistant Professor of Ophthalmology.
Harry K. Iwamota, Ph.D., Assistant Professor of Pharmacology.
H. Vernon Langeluttig, M.D., Assistant Professor of Medicine.[2]
John E. Legge, M.D., Assistant Professor of Medicine.[3]
Philip L. Lerner, Assistant Professor of Neurology.[2]
Hans W. Loewald, Assistant Professor of Psychiatry.[6]
John F. Lutz, A.B., M.D., Assistant Professor of Histology.
Stanley H. Macht, B.S., M.D., Assistant Professor of Roentgenology.
Howard B. Mays, M.D., Assistant Professor of Genito-Urinary Surgery and Instructor in Pathology.
Zachariah Morgan, M.D., Assistant Professor of Gastro-Enterology.
Samuel Morrison, A.B., M.D., Assistant Professor of Gastro-Enterology.
Harry M. Murdock, B.S., M.D., Assistant Professor of Psychiatry.
George McLean, M.D., Assistant Professor of Medicine.
Alfred T. Nelson, M.D., Assistant Professor of Anaesthesiology.[6a]
James W. Nelson, M.D., Assistant Professor of Surgery.
M. Alexander Novey, A.B., M.D., Assistant Professor of Obstetrics.
Dexter L. Reimann, B.S., M.D., Assistant Professor of Pathology,[7] Assistant in Medicine.[8]
I. O. Ridgely, M.S., M.D., Assistant Professor of Surgery.
Harry M. Robinson, Jr., B.S., M.D., Assistant Professor of Dermatology, Associate in Medicine.[2]
John E. Savage, B.S., M.D., Assistant Professor of Obstetrics.

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[6] Promoted January 1, 1947.
[6a] Appointed September 1, 1946.
[7] Appointed April 1, 1947.

Richard T. Shackelford, A.B., M.D., Assistant Professor of Surgery.
Isadore A. Siegel, A.B., M.D., Assistant Professor of Obstetrics.
Edward P. Smith, M.D., Ph.G., Assistant Professor of Gynecology.
R. Dale Smith, Ph.D., Assistant Professor of Gross Anatomy.
Sol Smith, M.D., Assistant Professor of Medicine.[2]
John H. Traband, M.D., Assistant Professor of Pediatrics.[3]
J. Ridgeway Trimble, M.D., Assistant Professor of Surgery.
John A. Wagner, B.S., M.D., Assistant Professor of Pathology[7] and Neuropathology,[1] Assistant in Medicine.
Philip S. Wagner, M.D., Assistant Professor of Psychiatry.[8]
W. Wallace Walker, M.D., Assistant Professor of Surgery and Surgical Anatomy.
Glenn S. Weiland, Ph.D., Assistant Professor of Biological Chemistry.[9]
Theodore E. Woodward, M.D., Assistant Professor of Medicine.[1]
Asa D. Young, M.D., Assistant Professor of Roentgenology.

ASSOCIATES

Conrad B. Acton, M.D., Associate in Medicine.[2]
Marie A. Andersch, Ph.D., Associate in Medicine.[2]
Margaret B. Ballard, M.D., Associate in Obstetrics.
Donald J. Barnett, M.D., Associate in Roentgenology.[2]
Eugene S. Bereston, A.B., M.D., Associate in Dermatology.[2]
Dudley P. Bowe, A.B., M.D., Associate in Obstetrics.[3]
Kenneth B. Boyd, A.B., M.D., Associate in Gynecology and Assistant in Obstetrics.
M. Paul Byerly, M.D., Associate in Medicine.[1]
Beverley C. Compton, A.B., M.D., Associate in Gynecology.
W. A. H. Councill, M.D., Associate in Genito-Urinary Surgery.
E. Eugene Covington, M.D., Associate in Surgical Anatomy and Surgery.
Francis G. Dickey, M.D., Associate in Medicine.[2]
D. McClelland Dixon, M.D., Associate in Obstetrics and Instructor in Pathology.
John C. Dumler, B.S., M.D., Associate in Gynecology.
William W. Elgin, M.D., Associate in Psychiatry.
J. J. Erwin, M.D., Associate in Gynecology.
Houston Everett, M.D., Associate in Gynecology.
L. K. Fargo, M.D., Associate in Genito-Urinary Surgery.
William L. Fearing, M.D., Associate in Neurology.[2]
Jerome Fineman, M.D., Instructor in Pediatrics.[2]
Samuel L. Fox, M.D., Associate in Rhinology, Laryngology and Otology.[2]
Irving Freeman, M.D., Associate in Medicine.[2]
George Govatos, A.B., M.D., Associate in Surgery.
Raymond F. Helfrich, A.B., M.D., Associate in Surgery.
W. Grafton Herspberger, M.D., Associate in Medicine.[2]
John T. Hibbitts, M.D., Associate in Gynecology.
John F. Hogan, M.D., Associate in Genito-Urinary Surgery.
Z. Vance Hooper, M.D., Associate in Gastro-Enterology.
Clewell Howell, B.S., M.D., Associate in Pediatrics.
Meyer W. Jacobson, M.D., Associate in Medicine.[2]
Joseph V. Jerardi, B.S., M.D., Associate in Surgery.

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[8] Promoted September 10, 1946.
[9] Appointed September 1, 1947.

D. Frank Kaltreider, A.B., M.D., Associate in Obstetrics.
Arthur Karfgin, B.S., M.D., Associate in Medicine.[2]
Fayne A. Kayser, M.D., Associate in Rhinology and Laryngology.[2]
Joseph I. Kemler, M.D., Associate in Ophthalmology.
F. Edwin Knowles, Jr., M.D., Associate in Ophthalmology.
Frederick R. Kyper, M.D., D.Sc., Associate in Rhinology, Laryngology,[2] and Bronchoscopy,[1] Instructor in Otology.[1]
C. Edward Leach, M.D., Associate in Medicine.[2]
Samuel Legum, M.D., Associate in Medicine.[2]
Luther E. Little, M.D., Associate in Surgery.
Ephraim T. Lisansky, M.D., Associate in Medicine.[2]
H. Edmund Levin, B.S., M.D., Associate in Bacteriology, Instructor in Medicine.[3]
G. Bowers Mansdorfer, B.S., M.D., Associate in Pediatrics.
I. H. Maseritz, M.D., Associate in Orthopaedic Surgery.[2]
Lyle J. Millan, M.D., Associate in Genito-Urinary Surgery.
Frank K. Morris, A.B., M.D., Associate in Gynecology, Instructor in Obstetrics.
W. Raymond McKenzie, M.D., Associate in Rhinology and Laryngology.
Hugh B. McNally, B.S., M.D., Associate in Obstetrics.
Frank N. Ogden, M.D., Associate in Biological Chemistry.[3]
Robert A. Reiter, M.D., Associate in Medicine.[2]
Samuel T. R. Revell, M.D., Associate in Medicine.[2]
William F. Rienhoff, Jr., M.D., Associate in Surgery.
Henry L. Rigdon, M.D., Associate in Surgery,[2] Assistant in Surgical Anatomy.[1]
Kathyrn L. Schultz, M.D., Associate in Psychiatry.[6]
Theodore A. Schwartz, M.D., Associate in Rhinology and Laryngology.[2]
William M. Seabold, A.B., M.D., Associate in Pediatrics.
Lawrence M. Serra, M.D., Associate in Medicine.[2]
William B. Settle, M.D., Associate in Surgery, Instructor in Surgical Anatomy.
A. Albert Shapiro, B.S., M.D., Associate in Dermatology.[2]
Arthur G. Siwinski, A.B., M.D., Associate in Surgery.
Benedict Skitarelic, A.B., M.D., Associate in Pathology,[2] Assistant in Medicine.[3]
E. H. Tonolla, M.D., Associate in Medicine.
William K. Waller, M.D., Associate in Medicine.[2]
Gibson J. Wells, M.D., Associate in Pediatrics.[2]
Austin H. Wood, M.D., Associate in Genito-Urinary Surgery.
Israel Zeligman, A.B., M.D., Associate in Dermatology.[2]

LECTURERS

Jonas Friedenwald, M.A., M.D., Lecturer in Ophthalmic Pathology.
Charles R. Goldsborough, M.A., M.D., Lecturer in Medicine.[3]
Leonard Karel, Ph.D., Lecturer in Pharmacology.[1]
Stephen Krop, Ph.D., Lecturer in Pharmacology.[1]
Leslie B. Hohman, M.D., Lecturer in Psychiatry.[3]
Joseph M. Miller, M.D., Lecturer in Surgery.
Myron G. Tull, A.B., M.D., M.P.H., Lecturer in Hygiene and Public Health.[3]

INSTRUCTORS

A. Russell Anderson, M.D., Instructor in Psychiatry.
Leon Ashman, M.D., Instructor in Medicine.[2]

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[6] Promoted September 10, 1946.

Carl E. Bailey, D.D.S., Instructor in Prosthetic Dentistry, School of Dentistry.
John Z. Bowers, B.S., M.D., Instructor in Medicine.[2]
Harry C. Bowie, B.S., M.D., Instructor in Surgery and Surgical Anatomy.
Thomas S. Bowyer, A.B., M.D., Instructor in Gynecology and Assistant in Obstetrics.
George H. Brouillet, B.S., M.D., Instructor in Surgery.
Ann Virginia Brown, A.B., Instructor in Biological Chemistry.
Samuel H. Bryant, A.B., D.D.S., Instructor in Oral Diagnosis, School of Dentistry.
Henry F. Buettner, M.D., Instructor in Bacteriology.[6]
Harold H. Burns, M.D., Instructor in Surgery.
Lucile J. Caldwell, M.D., Instructor in Dermatology.
Timothy A. Callahan, M.D., Instructor in Medicine.[1]
Mary E. Chinn, A.B., Instructor in Physiology.
Ernest I. Cornbrooks, Jr., A.B., M.D., Instructor in Gynecology.
Stuart G. Coughlan, B.S., M.D., Instructor in Surgery.
J. G. N. Cushing, M.D., Instructor in Psychiatry.
John R. Davis, M.D., Instructor in Medicine.[2]
B. Matthew Debuskey, M.D., Instructor in Pediatrics.[3]
W. Allen Deckert, A.B., M.D., Instructor in Gynecology and Assistant in Surgery.
William K. Diehl, B.S., M.D., Instructor in Gynecology.
Everett S. Diggs, B.S., M.D., Instructor in Gynecology.
Ernest S. Edlow, A.B., M.D., Instructor in Gynecology.
Philip D. Flynn, M.D., Instructor in Medicine.
William L. Garlick, A.B., M.D., Instructor in Surgery.
Jason H. Gaskel, M.D., Instructor in Orthopaedic Surgery.[2]
Samuel J. Hankin, M.D. Instructor in Medicine.[2]
E. M. Hanrahan, A.B., M.D., Instructor in Surgery.
Alvin J. Hartz, A.B., M.D., Instructor in Medicine.[1]
Mary L. Hayleck, M.D., Instructor in Pediatrics.
Robert F. Healy, M.D., Instructor in Surgery.
William G. Helfrich, B.S., M.D., Instructor in Medicine.
Benjamin Highstein, M.D., Instructor in Dermatology.
F. A. Holden, M.D., Instructor in Ophthalmology.
Mark B. Hollander, M.D., Instructor in Dermatology.
Helen A. Horn, A.B., M.D., Instructor in Pathology.[4]
Calvin Hyman, M.D., Instructor in Surgery.
Conrad L. Inman, D.D.S., Consulting Dentist, School of Dentistry.
Benjamin H. Isaacs, A.B., M.D., Instructor in Rhinology and Laryngology.
William R. Johnson, M.D., Instructor in Surgery.
Edward S. Kallins, B.S., M.D., Instructor in Medicine.
William H. Kammer, Jr., A.B., M.D., Instructor in Medicine.[2]
Clyde F. Karns, B.S., Instructor in Surgery.
Lester N. Kolman, M.D., Instructor in Dermatology.
A. Kremen, A.B., M.D., Instructor in Ophthalmology.
Louis J. Kroll, A.B., M.D., Instructor in Medicine.[2]
Milton C. Lang, M.D., Instructor in Ophthalmology.
Arnold F. Lavenstein, Instructor in Pediatrics.
Kurt Levy, M.D., Instructor in Medicine.
Joseph H. Marshall, M.D., Instructor in Psychiatry.[6a]

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[6] Resigned September 27, 1946.
[6a] Promoted September 10, 1946.

Karl F. Mech, B.S., M.D., Instructor in Gross Anatomy,[7] and Pathology.[2]
Israel P. Meranski, B.S., M.D., Instructor in Pediatrics.[2]
R. B. Mitchell, Jr., B.S., M.D., Instructor in Medicine.[3]
J. Duer Moores, B.S., M.D., Instructor in Surgery.
J. Huff Morrison, M.D., Instructor in Obstetrics.[8]
S. Edwin Muller, M.D., Instructor in Medicine.[2]
Joseph E. Muse, Jr., B.S., M.D., Instructor in Medicine.[2]
Ruth Musser, M.S., Instructor in Pharmacology.
John A. Myers, M.E.E., M.D., Instructor in Medicine, Assistant in Gastro-Enterology.
Francis J. McLaughlin, M.D., Instructor in Psychiatry.
Samuel Novey, M.D., Instructor in Psychiatry.
M. Paul Padget, M.D., Instructor in Medicine.
William A. Parr, M.D., Instructor in Otology.
Richard H. Pembroke, Jr., A.B., M.D., Instructor in Psychiatry.[6]
Eugene L. Pessagno, A.B., D.D.S., Instructor in Operative Surgery, School of Dentistry.
Leslie H. Pierce, B.S., M.D., Instructor in Medicine.[3]
Samuel E. Proctor, A.B., M.D., Instructor in Surgery.[1]
Herbert E. Reifschneider, A.B., M.D., Instructor in Surgery and Surgical Anatomy.
Daniel R. Robinson, M.D., Instructor in Surgery.
Seymour W. Rubin, M.D., Instructor in Pathology.[2]
John F. Schaefer, B.S., M.D., Instructor in Surgery.
Robert C. Sheppard, M.D., Instructor in Surgery.
M. S. Shiling, A.B., M.D., D.Sc., Instructor in Medicine.[3]
Albert J. Shochat, B.S., M.D., Instructor in Gastro-Enterology.
Harry A. Teitelbaum, B.S., M.D., Assistant in Neurology.[3]
David Tenner, M.D., Instructor in Medicine.
Lewis C. Toomey, D.D.S., Instructor in Oral Surgery, School of Dentistry.[1]
Wilfred H. Townshend, Jr., A.B., M.D., Instructor in Medicine.[2]
William D. VandeGrift, M.D., Instructor in Pathology.
Harold L. Vyner, M.D., Instructor in Psychiatry.[7a]
Frederick J. Vollmer, B.S., M.D., Instructor in Medicine.[2]
Hugh G. Whitehead, M.D., Instructor in Medicine.[3]
Milton J. Wilder, M.D., Instructor in Orthopaedic Surgery.
Daniel Wilfson, Jr., A.B., M.D., Instructor in Medicine.[2]

ASSISTANTS

J. Warren Albrittain, B.S., M.D., Assistant in Obstetrics.[8]
Harry McB. Beck, M.D., Assistant in Gynecology.
Robert Z. Berry, A.B., M.D., Assistant in Rhinology and Laryngology,[1] Assistant in Surgery.[3]
Henry A. Briele, M.D., Assistant in Surgery.[3]
Joseph G. Bird, A.B., M.D., Assistant in Pharmacology.[1]
William J. Bryson, A.B., M.D., Assistant in Pathology.[1]
A. V. Buchness, A.B., M.D., Assistant in Surgery.

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[6] Appointed January 1, 1947.
[7] Appointed September 5, 1946.
[7a] Appointed September 10, 1946.
[8] Appointed May 1, 1947.

Paul E. Carliner, B.S., M.D., Assistant in Medicine.[3]
John W. Chambers, M.D., Assistant in Surgery, and Neurological Surgery.[1]
L. T. Chance, M.D., Assistant in Surgery.
Robert F. Chenowith, M.D., Assistant in Surgery.[3]
Joseph M. Cordi, M.D., Assistant in Pediatrics.[1]
Samuel H. Culver, M.D., Assistant in Surgery.
Raymond M. Cunningham, A.B., M.D., Assistant in Surgery, and Proctology.[1]
Preston R. Cutler, A.B., M.D., Assistant in Surgery.
Edwin O. Daue, Jr., B.S., M.D., Assistant in Surgery.[3]
Nachman Davidson, A.B., M.D., Assistant in Medicine.[3]
E. Hollister Davis, A.B., M.D., Assistant in Anaesthesia.
George H. Davis, M.D., Assistant in Obstetrics.
John B. DeHoff, M.D., Assistant in Medicine.[1]
William A. Dodd, M.D., Assistant in Gynecology.
Charles H. Doeller, Jr., A.B., M.D., Assistant in Obstetrics and Gynecology.
William C. Duffy, A.B., M.D., Assistant in Gynecology.
William C. Dunnigan, A.B., M.D., Assistant in Surgery.
Morris A. Fine, M.D., Assistant in Medicine and Genito-Urinary Surgery.
Herbert M. Foster, M.D., Assistant in Surgery.[3]
Audrey M. Funk, A.B., Assistant in Medicine.
L. Calvin Gareis, B.S., M.D., Assistant in Obstetrics.[1]
William R. Geraghty, B.S., M.D., Assistant in Surgery.
Robert L. Gibbs, A.B., M.D., Assistant in Medicine.[3]
H. L. Granoff, A.B., M.D., Assistant in Gynecology.
William H. Grenzer, M.D., Assistant in Medicine.[3]
Joseph B. Gross, B.S., M.D., Assistant in Medicine.
John S. Haines, M.D., Assistant in Genito-Urinary Surgery.[1]
Donald B. Hebb, M.D., Assistant in Surgery and Proctology.[6]
Oscar Hartman, B.S., M.D., Assistant in Obstetrics.[3]
John S. Haught, M.D., Assistant in Gynecology.
Thomas A. Hedrick, M.D., Assistant in Pediatrics.[3]
L. Ann Hellen, B.S., Assistant in Medicine.
James W. Hendrick, M.D., Assistant in Surgery, and Plastic Surgery.[1]
John H. Hirschfeld, M.D., Assistant in Rhinology and Laryngology.
John V. Hopkins, M.D., Assistant in Orthopaedic Surgery.
Rollin C. Hudson, M.D., Assistant in Medicine.
Jaroslav Hulla, M.D., Assistant in Obstetrics.[3]
William C. Humphries, M.D., Assistant in Rhinology and Laryngology.[3]
Jacob R. Jensen, B.S., M.D., Assistant in Obstetrics.[3]
Hugh Jewett, M.D., Assistant in Genito-Urinary Surgery.
Harry F. Kane, M.D., Assistant in Gynecology.
Melvin D. Kappelman, B.S., M.D., Assistant in Obstetrics.
James R. Karns, B.S., M.D., Assistant in Medicine.
Lawrence Katzenstein, M.D., Assistant in Medicine.[3]
Lauriston L. Keown, A.B., M.D., Assistant in Pediatrics.[3]
Irvin P. Klemkowski, M.D., Assistant in Obstetrics.[3]
Charles W. Knerler, M.D., Assistant in Medicine.[3]
Schuyler G. Kohn, B.S., M.D., Assistant in Obstetrics.

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[6] Appointed November 1, 1946.

Alfred S. Lederman, Assistant in Gastro-Enterology.
Frank E. Leslie, A.B., M.D., Assistant in Medicine.[1]
Etta C. Link, B.S., M.D., Assistant in Pediatrics.[1]
V. Harwood Link, A.B., M.D., Assistant in Dermatology.
J. Douglas Lockard, M.D., Assistant in Medicine.[1]
F. Ford Loker, B.S., M.D., Assistant in Surgery.
H. Pearce Maccubbin, B.S., M.D., Assistant in Medicine.[3]
John W. Machen, M.D., Assistant in Neurology.[3]
Helen I. Maginnis, M.D., Assistant in Gynecology.
W. Kenneth Mansfield, Jr., M.D., Assistant in Obstetrics.
Charles B. Marek, M.D., Assistant in Gynecology.
A. Robert Marks, A.B., M.D., Assistant in Pediatrics.
Maxwell L. Mazer, M.D., Assistant in Obstetrics.
Robert B. Mearns, M.D., Assistant in Medicine.[3]
George C. Medairy, M.D., Assistant in Neurology.[3]
William A. Mitchell, M.D., Assistant in Obstetrics.
John H. Morrison, B.S., M.D., Assistant in Obstetrics.
Howard B. McElwain, M.D., Assistant in Surgery.
John W. Osborne, M.D., Assistant in Medicine.[1]
Frank J. Otanasek, M.D., Assistant in Neurological Surgery.[1]
Margaret Virginia Palmer, A.M., M.D., Assistant in Medicine.[3]
Patrick C. Phelan, Jr., A.B., M.D., Assistant in Surgery.
Ross Z. Pierpont, M.D., Assistant in Surgery,[3] and Surgical Anatomy.
‡Samuel E. Proctor, A.B., M.D., Assistant in Surgery.
J. Emmett Queen, A.B., M.D., Assistant in Medicine.[3]
William T. Raby, B.S., M.D., Assistant in Pathology.[1]
Frederick M. Reese, A.B., M.D., Assistant in Ophthalmology.
John O. Robben, B.S., M.D., Assistant in Plastic Surgery.[3]
T. Edgie Russell, Jr., B.S., M.D., Assistant in Obstetrics.
William J. Rysanek, Jr., M.D., Assistant in Gynecology.
Clarence P. Scarborough, M.D., Assistant in Plastic Surgery.
W. J. Schmitz, M.D., Assistant in Pediatrics.[3]
Earle S. Scott, M.D., Assistant in Pediatrics.[3]
J. King B. E. Seegar, Jr., A.B., M.D., Assistant in Obstetrics.
Kenneth C. Sharrets, A.B., M.D., Assistant in Surgery.[1]
Joseph C. Sheehan, B.S., M.D., Assistant in Gynecology.
E. Roderick Shipley, A.B., M.D., Assistant in Surgery.
George Silverton, A.B., M.D., Assistant in Medicine.[3]
Jerome Snyder, B.S., M.D., Assistant in Ophthalmology.
June Linda Snyder, A.B., Assistant in Physiology.
Samuel Snyder, M.D., Assistant in Medicine.
Walter Spurrier, M.D., Assistant in Pediatrics.[1]
Edwin H. Stewart, Jr., M.D., Assistant in Surgery and Plastic Surgery.[1]
William J. Supik, M.D., Assistant in Proctology.[1]
Adam Swiss, M.D., Assistant in Medicine.[1]
Raymond K. Thompson, B.S., M.D., Assistant in Neurological Surgery.[1]
T. J. Touhey, M.D., Assistant in Surgery.
W. H. Triplett, M.D., Assistant in Medicine.[3]
Robert B. Tunney, A.B., M.D., Assistant in Gynecology.
Stephen J. Van Lill, III, A.B., M.D., Assistant in Medicine.[1]
Frederick J. Vollmer, B.S., M.D., Assistant in Medicine.

Sholom O. Waife, M.D., Assistant in Medicine, [1]
William Earl Weeks, M.D., Assistant in Pediatrics.
J. Carlton Wich, B.S., M.D., Assistant in Pediatrics.
Albert R. Wilkerson, M.D., Assistant in Surgery.
Thomas L. Worsley, M.D., Assistant in Medicine.[1]
Isaac C. Wright, A.B., M.D., Assistant in Pathology.[1]
Howard L. Zupnik, M.D., Assistant in Surgery.

FELLOWS

Frederick K. Bell, Ph.D., U. S. Pharmacopoeia Fellow.
Joseph G. Bird, A.B., M.D., Fellow in Pharmacology.[1]
R. Adams Cowley, M.D., Fellow in Surgery.[1]
Robert M. H. Crosby, M.D., Fellow in Neurological Surgery.[1]
Everett S. Diggs, B.S., M.D., Hitchcock Fellow in Gynecology.[1]
Nancy Jane Ferneyhough—Fellow in Art as Applied to Medicine.[3]
Elaine Gaby, A.B., Emerson Fellow in Pharmacology.
Richard D. Grill, Fellow in Art as Applied to Medicine.
James W. Hendrick, M.D., National Cancer Institute Trainee.[1]
Mary J. Sauerwald, A.B., Emerson Fellow in Pharmacology.
George W. Smith, M.D., Hitchcock Fellow in Neurological Surgery.[1]
Carl Christian Stein—Fellow in Art as Applied to Medicine.[3]
Edwin H. Stewart, Jr., M.D., National Cancer Institute Trainee.[1]
Edward B. Truitt, B.S., Markle Fellow in Pharmacology.[6]
Hugh D. Vazzana, M.D., Fellow in Roentgenology.[1]

UNIVERSITY HOSPITAL

EXECUTIVE COMMITTEE OF THE STAFF

H. Boyd Wylie, *Acting Dean*,
Harold A. Sayles, *Acting Superintendent*, } *Ex officio*

	Alternates
Beverly C. Compton, *Chairman*	
W. Houston Toulson, *Vice Chairman*	
Maurice C. Pincoffs	T. Nelson Carey
Arthur M. Shipley	Charles Reid Edwards
Louis H. Douglass	J. G. M. Reese
J. Mason Hundley, Jr.	
Clyde A. Clapp	Henry F. Graff
Edward A. Looper	Thomas R. O'Rourk
Walter L. Kilby	Charles N. Davidson
C. Loring Joslin	J. Edmund Bradley
Allen Fiske Voshell	
Frederick C. Dye	
Hugh R. Spencer	
George H. Yeager	
William M. Seabold, *Secretary*	

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[6] December 1, 1946 to June 30, 1947.

UNIVERSITY HOSPITAL STAFF

Harold A. Sayles, *Acting Superintendent*

Physician-in-Chief	Maurice C. Pincoffs
Physicians	Walter A. Baetjer Thomas P. Sprunt John E. Legge Louis A. M. Krause G. Carroll Lockard Joseph E. Gichner William H. Smith William S. Love, Jr. T. Nelson Carey
Gastro-Enterologist	Zachariah R. Morgan
Neurologists	Irving J. Spear Leon Freedom
Psychiatrists	Ross McC. Chapman Ralph P. Truitt Harry M. Murdock
Pediatricians	C. Loring Joslin Albert Jaffe J. Edmund Bradley A. H. Finkelstein William M. Seabold
Pathologist	Hugh R. Spencer
Surgeon-in-Chief	Arthur M. Shipley
Surgeons	Charles Reid Edwards Thomas B. Aycock
Neurological Surgeons	Charles Bagley, Jr. Richard G. Coblentz James G. Arnold, Jr.
Laryngologists	Edward A. Looper Franklin B. Anderson Thomas R. O'Rourk
Proctologists	Monte Edwards Thurston R. Adams
Orthopaedic Surgons	Allen Fiske Voshell Moses Gellman Compton Riely Henry F. Ullrich
Genito-Urinary Surgeons	W. Houston Toulson W. A. H. Councill Lyle J. Millan Howard B. Mays
Dental Surgeon-in-Chief	Brice M. Dorsey
Dentists	Vernon D. Kaufman Samuel H. Bryant Edward C. Dobbs Lewis C. Toomey[1] Eugene L. Pessagno

Roentgenologists	Walter L. Kilby Charles N. Davidson Donald J. Barnett
Dermatologist	Harry M. Robinson, Sr.
Assistant Dermatologists	Francis A. Ellis[1] Harry M. Robinson, Jr.[1]
Bronchoscopist	Edward A. Looper
Associate Bronchoscopist	Frederick T. Kyper[1]
Assistant Bronchoscopist	John H. Hirschfeld[1]
Otologist	Thomas R. O'Rourk
Anaesthesiologists	Frederick C. Dye Alfred T. Nelson
Obstetricians	Louis H. Douglass Isadore A. Siegel J. G. M. Reese John E. Savage Hugh B. McNally
Ophthalmologists	Clyde A. Clapp Henry F. Graff F. Edwin Knowles, Jr.
Gynecologists	J. Mason Hundley, Jr. Leo Brady Beverley C. Compton
Oncologists	J. Mason Hundley, Jr. Grant E. Ward

ANNUAL HOSPITAL APPOINTMENTS

The following annual appointments are made to the University Hospital:

Three Residents in Medicine
Seven Residents in Surgery
One Resident in Neurosurgery
Three Residents in Gynecology
Four Residents in Obstetrics
Two Residents in Pediatrics
Two Residents in Roentgenology
Twenty-Four Rotating Interns

UNIVERSITY HOSPITAL RESIDENT AND INTERN STAFF

July 1, 1947 to June 30, 1948

RESIDENT STAFF

A. Maynard Bacon, Jr., B.S., M.D., *Assistant Resident in Pediatrics*
[1]Ruth W. Baldwin, B.S., M.D., *Assistant Resident in Pediatrics*[1]
Robert E. Bauer, A.B., M.D., *Assistant Resident in Medicine*
William A. Burke, B.S., M.D., *Resident in Pediatrics*
William L. Byerly, B.S., M.D., *Assistant Resident in Surgery*[9]
[2]Eugene C. Conner, M.D., *Assistant Resident in Anaesthesiology*
Sarah Cook, A.B., M.D., *Assistant Resident in Medicine*
Richard J. Cross, B.S., M.D., *Assistant Resident in Eye, Ear, Nose and Throat*
Walter L. Crouch, B.S., M.D., *Assistant Resident in Pediatrics*
Edwin O. Daue, Jr., B.S., M.D., *Resident in Surgery*

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[1] Until October 31, 1947.

[2] Beginning about September 15, 1947.

John B. Doenges, A.B., M.D., *Assistant Resident in Medicine*
James L. Eavey, B.S., M.D., *Assistant Resident in Gynecology*
John F. Gayle, M.D., *Assistant Resident in Gynecology (assigned to Obstetrics)*
James H. Graves, B.S., M.B., M.D., *Assistant Resident in Obstetrics (assigned to Gynecology)*
Ernest G. Guy, B.S., M.D., *Resident in Medicine*
[3]Gunyon Harrison, M.D., *Assistant Resident in Pediatrics*
Charles W. Hawkins, M.D., *Assistant Resident in Surgery*
C. Hal Ingram, A.B., M.D., *Assistant Resident in Surgery*
Erwin R. Jennings, A.B., M.D., *Assistant Resident in Surgery*
Theodore Kardash, B.S., M.D., *Resident in Gynecology*
Joseph M. Keith, A.B., M.D., *Assistant Resident in Obstetrics*
Robert E. Lartz, B.S., M.D., *Assistant Resident in Obstetrics*
Clarence W. Legerton, Jr., M.D., *Assistant Resident in Medicine*
Lorman L. Levinson, A.B., M.D., *Resident in Obstetrics*
William D. Lynn, A.B., M.D., *Assistant Resident in Surgery*
Louis O. Manganiello, A.B., M.D., *Assistant Resident in Neurosurgery*
Clarence Martin, M.D., *Assistant Resident in Obstetrics*
George Y. Massenburg, M.D., *Assistant Resident in Surgery*
Edward B. McGovern, B.S., M.D., *Assistant Resident in Anaesthesiology*
E. Thornton Pfeil, B.S., M.D., *Resident in Neurosurgery*
James J. Range, A.B., M.D., *Assistant Resident in Roentgenology*
Howard L. Seabright, M.D., *Assistant Resident in Pediatrics*
Edwin L. Seigman, A.B., M.D., *Resident in Roentgenology*
[4]Cecil W. Shafer, B.S., M.D., *Assistant Resident in Anaesthesiology*
Robert L. Stone, A.B., M.D., *Assistant Resident in Gynecology*
James A. Vaughn, Jr., A.B., M.D., *Assistant Resident in Anaesthesiology*
James H. Walker, A.B.. M.D., *Assistant Resident in Surgery*
David R. Will, M.D., *Assistant Resident in Surgery*
Joseph B. Workman, A.B., M.D., *Assistant Resident in Medicine*

INTERNS

John F. Benson, B.S., M.D.
Frank E. Brumback, M.D.
Robert C. Duvall, Jr., M.D.
John E. Evans, Jr., B.S., M.D.
William A. Gakenheimer, M.D.
Benjamin M. Gold, B.S., M.D.
Robert R. Hahn, M.D.
Howard E. Hall, M.D.
Calvin B. Hearne, M.D.
John A. Hightower, M.D.
Robert C. Hunter, B.S., M.D.
Benjamin L. Jamison, A.B., M.D.
Arlie R. Mansberger, Jr., M.D.
Richard A. Neubauer, B.S., M.D.
David L. Phillips, A.B., M.D.
John B. Piggott, B.S., M.D.
Jack H. Powell, Jr., A.B., M.D.
John H. Rosser, B.S., M.D.
Sidney H. Sachs, M.D.
Joseph Shear, B.S., M.D.
Harold N. Taylor, A.B., M.D.
John P. White, III, M.D.
James C. Yockey, B.A., M.D.
Edgar W. Young, Jr., B.A., M.D.

DENTAL INTERNS

William J. Coleman, Jr., D.D.S.
William D. Hartsock, B.A., D.D.S.

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[3] July, August, and September, 1947.
[4] Beginning August 1, 1947.

UNIVERSITY HOSPITAL DISPENSARY STAFF

EMMA WINSHIP, R.N.
Dispensary Director

DISPENSARY COMMITTEE

GEORGE H. YEAGER, *Chairman*
EMMA WINSHIP, R.N., *Secretary*

ALLEN FISKE VOSHELL
BEVERLEY C. COMPTON
A. H. FINKELSTEIN
LEWIS P. GUNDRY
HOWARD B. MAYS
D. FRANK KALTREIDER

Chief of Medical Clinic	LEWIS P. GUNDRY
Assistant Chief of Medical Clinic	M. PAUL BYERLY[1]
Physicians	M. A. FINE SAMUEL J. HANKIN LEON ASHMAN[1] JOHN B. DEHOFF[1] LEON A. KOCHMAN[1] THOMAS H. MAGNESS[1] ELIZABETH D. SHERRILL[1] CHARLES H. WILLIAMS[1] CHARLES R. GOLDSBOROUGH[3] MEYER W. JACOBSON[3] H. EDMUND LEVIN[3] THOMAS P. SPRUNT[3] G. CARROLL LOCKARD[3] MILTON S. SACKS[3] LESLIE HARRELL PIERCE[3]
Chief of Gastro-Enterology Clinic	FRANCIS G. DICKEY[1]
Assistant Chiefs of Gastro-Enterology Clinic	Z. VANCE HOOPER FRANCIS G. DICKEY[3]
Assistant Gastro-Enetrologists	ALBERT J. SHOCHAT ALFRED S. LEDERMAN SAMUEL MORRISON[3] CHARLES V. SEVCIK[3]
Chief of Neurology Clinic	LEON FREEDOM
Assistant Neurologists	WILLIAM L. FEARING HARRY A. TEITELBAUM[3]
Chief of Psychiatric Clinic	RALPH P. TRUITT
Clinical Director	H. W. NEWELL[1]
Assistant Psychiatrists	ALICE J. ROCKWELL HANS W. LOEWALD KATHRYN L. SCHULTZ HAROLD L. VYNER HARRY GOLDSMITH[3] JOSEPH MARSHALL[3] H. ROBERT BLANK[3] SIDNEY BERMAN[3] MABEL WILKINS[3] HELEN ARTHUR[3]

Chief of Chest Clinic	MEYER D. JACOBSON[1]
Assistant, Diseases of the Lungs	MANUEL LEVIN MEYER W. JACOBSON[3] JOSEPH E. MUSE, JR.[3]
Chief of Metabolism Clinic	W. GRAFTON HERSPERGER[1] LEWIS P. GUNDRY[3]
Assistants in Metabolism Clinic	LOUIS V. BLUM WILLIAM G. HELFRICH[3] WALTER E. KARFGIN[3] A. GOLDMAN[3] W. GRAFTON HERSPERGER[3]
Chief of Cardiovascular Clinic	C. EDWARD LEACH[1] WILLIAM S. LOVE, JR.[3]
Assistant Cardiologists	H. F. ERMSHAR WILFRED H. TOWNSHEND WILLIAM G. HELFRICH ROLLIN C. HUDSON STEPHEN J. VAN LILL, III[1] SAMUEL J. HANKIN[3] IRVING FREEMAN[3] HARRY M. ROBINSON, JR.[3] EPHRAIM T. LISANSKY[3] F. FREDERICK RUZICKA[3]
Chief of Allergy Clinic	H. M. BUBERT
Assistant Chief of Allergy Clinic	EDWARD S. KALLINS
Assistant Allergists	SAMUEL SNYDER FRANK V. MANIERI[3] S. EDWIN MULLER[3]
Allergy Clinic Technician	SELMA ROSENBERG
Director, Pediatric Clinic	A. H. FINKELSTEIN
Chief of Pediatric Clinic	SAMUEL S. GLICK
Assistant Pediatricians	JEROME FINEMAN JOSEPH R. MEYEROWITZ LOUIS V. BLUM ARNOLD F. LAVENSTEIN THOMAS E. WEEKS J. CARLTON WICH RAMSAY B. THOMAS[1] HOWARD GOODMAN[1] MELVIN N. BORDEN[1] KEACIL K. KRULEVITZ[1] ETTA C. LINK[1] LESTER KAPLAN[1] ISRAEL P. MERANSKI[3] WILLIAM M. SEABOLD[3] ALBERT JAFFE[3] LAURISTON L. KEOWN[3] GIBSON J. WELLS[3] EARLE S. SCOTT[3] A. ROBERT MARKS[3]

Chief of Endocrinology Clinic	John Z. Bowers[1]
Chief of Surgical Clinic	George H. Yeager
Assistant Surgeons	George H. Brouillet Stuart G. Coughlan William R. Johnson Samuel E. Proctor[1] Clarence P. Scarborough William B. Settle
Chief of Orthopaedic Surgery Clinic	Allen Fiske Voshell
Assistant Orthopaedic Surgeons	John V. Hopkins Moses Gellman Henry F. Ullrich Milton J. Wilder Everett D. Jones
Chief of Genito-Urinary Clinic	W. Houston Toulson
Assistant Genito-Urinary Surgeons	W. A. H. Councill John F. Hogan Lyle J. Millan Morris A. Fine[1] Howard B. Mays
Director of Dermatology Clinic	Harry M. Robinson, Sr.
Chief of Dermatology Clinic	Harry M. Robinson, Jr.
Assistant Dermatologists	Francis A. Ellis Eugene S. Bereston A. Albert Shapiro Lester N. Kolman Benjamin Highstein Lucile Caldwell V. Harwood Link R. C. V. Robinson[1] Alfred H. Dann[1] John M. Seigel[1] Israel Zeligman[1] Mark B. Hollander[3]
Chief of Rhinology and Laryngology Clinic	Benjamin S. Rich
Assistant Rhinologists and Laryngologists	Thomas R. O'Rourk M. Baylus Frederick T. Kyper Samuel L. Fox John H. Hirschfeld[1]
Chief of Proctology Clinic	Monte Edwards
Assistant Proctologists	Thurston R. Adams Donald B. Hebb[1] William J. Supik[1]
Chief of Gynecology Clinic	J. Mason Hundley, Jr.
Assistant Chief of Gynecology Clinic	Beverley C. Compton

Assistant Gynecologists	Thomas S. Bowyer John C. Dumler John T. Hibbitts Ernest I. Cornbrooks, Jr. William B. Settle Kenneth B. Boyd W. Allen Deckert Everett S. Diggs Helen I. Maginnis William K. Diehl John S. Haught
Female Cystoscopists	J. Mason Hundley, Jr. Beverley C. Compton William K. Diehl
Chief of Dental Clinic	Brice M. Dorsey
Assistant Chief of Dental Clinic	Vernon D. Kaufman
Assistant Dentists	Carl E. Bailey[3] Samuel H. Bryant Edward C. Dobbs Eugene L. Pessagno Lewis C. Toomey[1]
Chief of Obstetrical Clinic	D. Frank Kaltreider
Assistant Chiefs of Obstetrical Clinic	Margaret B. Ballard J. Huff Morrison
Assistant Obstetricians	Dudley P. Bowe[3] Maxwell L. Mazer Jaroslav Hulla[3] Hugh B. McNally Jacob R. Jensen[3] J. Warren Albrittain[3] D. McClelland Dixon W. Kenneth Mansfield, Jr. Thomas S. Bowyer L. Calvin Gareis Oscar Hartman[3] Melvin D. Kappelman William A. Mitchell J. K. B. E. Seegar T. Edgie Russell, Jr. Irvin P. Klemkowski[3] Kenneth B. Boyd
Oncology Clinic, Gynecological Division	J. Mason Hundley, Jr. Beverley C. Compton John C. Dumler William K. Diehl Ernest I. Cornbrooks, Jr. Everett S. Diggs

Oncology Clinic, Surgical Division	GRANT E. WARD E. EUGENE COVINGTON J. DUER MOORES ARTHUR G. SIWINSKI
Chief of Vascular Clinic	GEORGE H. YEAGER
Assistant Chief of Vascular Clinic	GEORGE H. BROUILLET
Medical Consultant-Vascular Clinic	LEWIS P. GUNDRY
Chief of Ophthalmology Clinic	HENRY F. GRAFF
Assistant Ophthalmologists	FRANK A. HOLDEN MILTON C. LANG JEROME SNYDER FREDERICK M. REESE
Directress, Occupational Therapy	MISS FRANCES F. LEWIS
Directress, Social Service	MISS MARY FITZPATRICK

UNIVERSITY HOSPITAL DISPENSARY REPORT

July 1, 1945–June 30, 1946

Departments	*New Cases*	*Old Cases*	*Tota*
Allergy	107	2,126	2,233
Cardiology	116	1,255	1,371
Cystoscopy	74	393	467
Dermatology	4,304	9,834	14,138
Diabetic	58	576	634
Ear, Nose & Throat	934	900	1,834
Eye	1,136	3,041	4,177
Gastro-Intestinal	240	897	1,137
Genito-Urinary	454	979	1,433
Gynecology	1,460	4,318	5,778
Hematology	6	139	145
Medicine	1,274	3,270	4,544
Neurology	182	610	792
Obstetrics	1,639	11,049	12,688
Occupational Therapy	108	1,392	1,500
Oncology	195	953	1,148
Orthopedic	1,171	2,507	3,678
Pediatrics	1,588	7,297	8,885
Physiotherapy	145	923	1,068
Plastic Surgery	8	14	22
Proctology	152	153	305
Psychiatry	113	377	490
Surgery	2,954	6,724	9,678
Tuberculosis	126	621	747
Total	18,544	60,348	78,892

MERCY HOSPITAL

BOARD OF GOVERNORS

Walter D. Wise, *Chairman*

Sister M. Veronica
Sister M. Celeste
Sister M. Mildred
Sister M. Philip
Sister M. Vincent
Sister M. Helen

Henry F. Bongardt
H. Raymond Peters
Maurice C. Pincoffs
Waitman F. Zinn
Thomas K. Galvin
Edward P. Smith
Elliott H. Hutchins

MERCY HOSPITAL STAFF

Surgeon-in-Chief	Walter D. Wise
Surgeons	Elliott H. Hutchins Charles F. Blake F. L. Jennings R. W. Locher Thomas R. Chambers D. J. Pessagno William F. Rienhoff N. Clyde Marvel Henry F. Bongardt
Neurological Surgeons	Charles Bagley, Jr. Richard B. Coblentz James D. Arnold, Jr. Frank J. Otenasek John W. Chambers[1]
Associate Surgeons	I. O. Ridgely James W. Nelson Howard B. McElwain Simon H. Brager John A. O'Connor Charles W. Maxson I. Ridgeway Trimble Richard T. Shackelford Raymond F. Helfrich
Assistant Surgeons	Julius Goodman S. Demarco, Jr. T. J. Touhey William N. McFaul, Jr. Meyer H. Zuravin Howard L. Zupnik Daniel R. Robinson Joseph V. Jerardi Wm. C. Dunnigan Harold H. Burns William L. Garlick John F. Schaeffer F. Ford Loker Patrick C. Phelan Kenneth C. Sharrets[1]

Plastic Surgeon	Edward A. Kitlowski Clarence P. Scarborough[1]
Consulting Ophthalmologist and Otologist	Harry Friedenwald
Ophthalmologists and Otologists	H. K. Fleck M. Raskin
Associate Ophthalmologists and Otologists	Joseph I. Kemler F. A. Pacienza
Assistant Ophthalmologists and Otologists	Joseph V. Jeppi F. Edwin Knowles, Jr.
Consulting Rhinologists and Laryngologists	W. Raymond McKenzie George W. Mitchell
Rhinologist and Laryngologist	Waitman F. Zinn
Associate Rhinologists and Laryngologists	Fayne A. Kayser Benjamin S. Rich Theodore A. Schwartz Birkhead Macgowan Benjamin H. Isaacs
Assistant Rhinologist and Laryngologist	Joseph V. Jeppi
Bronchoscopist	Waitman F. Zinn
Associate Bronchosopist	Fayne A. Kayser
Assistant Bronchoscopist	Theodore A. Schwartz
Proctologist	Charles F. Blake
Consulting Orthopaedic Surgeon	Albertus C. Cotton
Orhtopaedic Surgeon	H. L. Rogers
Associate Orthopaedic Surgeon	Henry F. Ullrich
Assistant Orthopaedic Surgeons	I. H. Maseritz J. H. Gaskel
Urologist	Kenneth D. Legge
Associate Urologists	Leon K. Fargo Francis W. Gillis
Dermatologists	Francis A. Ellis William D. Wolfe Eugene S. Bereston
Dentist	J. D. Fusco
Consulting Dentist	Conrad L. Inman
Consulting Physician	Maurice C. Pincoffs
Physician-in-Chief	H. Raymond Peters
Physicians	Harvey G. Beck Thomas P. Sprunt George McLean J. Sheldon Eastland Louis A. M. Krause Thomas C. Wolff

Associate Physicians	Hubert C. Knapp Bartus T. Baggott Wetherbee Fort John E. Legge T. Nelson Carey Sol Smith Hugh J. Welch
Assistant Physicians	S. A. Tumminello J. Howard Burns Earl L. Chambers K. W. Golley William H. Kammer S. Edwin Muller John R. Davis, Jr. J. Emmett Queen Philip D. Flynn Frederick J. Vollmer
Associate Gastro-Enterologists	R. Frederick Leitz Theodore H. Morrison Maurice Feldman
Assistant Gastro-Enterologist	Ephraim T. Lisansky
Pediatricians	Edgar B. Friedenwald Frederick B. Smith
Associate Pediatrician	G. Bowers Mansdorfer
Assistant Pediatricians	William M. Seabold Jerome Fineman O. Walter Spurrier Israel P. Meranski Edward L. Frey, Jr.
Neurologist and Psychiatrist	Andrew C. Gillis
Associate Neurologists and Psychiatrists	Harry Goldsmith Philip F. Lerner
Obstetrician-in-Chief	Edward P. Smith
Obstetricians	J. J. Erwin Thomas K. Galvin William S. Gardner Frank K. Morris Ernest S. Edlow Hugh B. McNally
Associate Obstetricians	William C. Duffy Charles H. Doeller, Jr.
Assistant Obstetricians	J. Howard Burns Harry F. Kane William A. Dodd Harry McB. Beck Joseph C. Sheehan William J. Rysanek, Jr. Robert B. Tunney

Consulting Gynecologist	William S. Gardner
Gynecologist-in-Chief	Thomas K. Galvin
Gynecologists	Edward P. Smith J. J. Erwin Frank K. Morris
Associate Gynecologists	Ernest S. Edlow George A. Strauss, Jr. H. L. Granoff
Assistant Gynecologists	Charles H. Doeller, Jr. William A. Dodd Harry McB. Beck William C. Duffy Joseph C. Sheehan William J. Rysanek, Jr. Harry F. Kane Robert B. Tunney
Pathologists	Walter C. Merkel Hugh R. Spencer
Clinical Pathologist	H. T. Collenberg
Clinical Biochemist	Charles E. Brambel
Technicians	Sister Paula Marie Eleanor Behr Charlotte Schwalm Lillian Marie Lawler Elizabeth Johnson Carmela E. Miceli
Consulting Radiologist	Albertus Cotton
Radiologist	Asa D. Young
Assistant Radiologist	E. Eugene Covington
Technicians	Sister Paula Marie Eleanor Behr Elizabeth Johnson Carmela E. Miceli Lillian Butler Betty Wolfram Virginia Schwarz Julietta Perez
Technician (X-ray)	Ruth L. Gephardt

ANNUAL HOSPITAL APPOINTMENTS

The following annual appointments are made to the Mercy Hospital:

Five Residents in Surgery
Four Residents in Medicine
Resident in Pathology
Resident in Rhinology
Three Residents in Gynecology and Obstetrics
Twelve Interns on Rotating Service

MERCY HOSPITAL RESIDENT AND INTERN STAFF

JULY 1. 1947–JUNE 30, 1948

RESIDENT STAFF

Michael L. DeVincentis, B.S., M.D., *Resident Surgeon*
William D. McClung, M.D., *Associate Resident Surgeon*
James G. Stegmaier, B.S., M.D., *Assistant Resident Surgeon*
Edward P. Smith, Jr., M.D., *Assistant Resident Surgeon*
David N. Sills, Jr., M.D., *Assistant Resident Surgeon*
Walter K. Spelsberg, B.S., M.D., *Resident Gynecologist*
Richard E. Hunter, A.B., M.D., *Resident Obstetrician*
William D. Gentry, Jr., M.D., *Assistant Resident Gynecologist and Obstetrician*
Richard A. Carey, B.S., M.D., *Resident Physician*
James J. Nolan, B.S., M.D., *Assistant Resident Physician*
Lawrence J. Knox, Jr., B.S., M.D., *Assistant Resident Physician*
Clarence E. McWilliams, Jr., A.B., M.D., *Assistant Resident Physician*
James J. Gerlach, A.B., M.D., *Resident Otolaryngologist*

RESIDENT INTERNS

Robert J. Johnson, M.D.
Harry B. Scott, M.D.
John G. Brickner III, B.S., M.D.
D. Otis Montgomery, A.B., M.D.
William F. Schnitzker, A.B., M.D.
Lane McD. Jernigan, B.S., M.D.
Elden H. Pertz, B.S., M.D.
Frank A. Faraino, B.S., M.D.
Henry V. Chase, B.S., M.D.
William R. Post, B.A., M.D.
Joseph F. Li Pira, B.S., M.D.

MERCY HOSPITAL DISPENSARY STAFF

Sister M. Agnesine, B.S., R.N. .... *Dispensary Director*

Supervisor of Surgical Clinic .... Raymond F. Helfrich

Assistant Supervisor of Surgical Clinic .... Harold H. Burns

Dispensary Surgeons	I. O. Ridgely H. F. Bongardt James W. Nelson I. Ridgeway Trimble Simon H. Brager S. Demarco, Jr. Richard T. Shackelford Howard L. Zupnik Daniel R. Robinson Joseph V. Jerardi William C. Dunnigan William L. Garlick John F. Schaefer F. Ford Loker Patrick C. Phelan Kenneth C. Sharrets[1]
Supervisor of Genito-Urinary Clinic	Kenneth D. Legge
Assistant Genito-Urinary Surgeons	L. K. Fargo Francis W. Gillis John S. Haines[1]
Supervisor of Orthopaedic Clinic	Harry L. Rogers
Orthopaedic Surgeons	Henry F. Ullrich I. H. Maseritz Jason H. Gaskel
Supervisors of Medical Clinic	H. Raymond Peters Maurice C. Pincoffs
Chiefs of Medical Clinic	Sol Smith S. Edwin Muller
Assistant Physicians	Milton Stein Irving Freeman Philip D. Flynn Frederick J. Vollmer William H. Kammer John R. Davis J. Emmett Queen Charles F. O'Donnell Joseph R. Guyther
Chief of Allergy Clinic	S. Edwin Muller
Chief of Cardiovascular Clinic	Thomas C. Wolff
Assistant Cardiologists	Leon Ashman Ephraim T. Lisansky
Chief of Metabolism Clinic	J. Sheldon Eastland
Assistant in Metabolism Clinic	
Gastro-Enterologist	Maurice Feldman
Assistant Gastro-Enterologist	Ephraim T. Lisansky
Chief of Pediatric Clinic	Edgar B. Friedenwald

Pediatricians	G. Bowers Mansdorfer Jerome Fineman George Bayley John W. Machen Israel T. Meranski O. Walter Spurrier Edward L. Frey, Jr.
Supervisor of Neurological and Psychiatric Clinic	Andrew C. Gillis
Neurologists and Psychiatrists	Harry Goldsmith Philip F. Lerner George C. Mediary
Supervisor of Dermatology Clinic	Francis A. Ellis
Dermatologists	William D. Wolfe Eugene S. Bereston R. C. V. Robinson[1]
Oncologist	James W. Nelson
Chief of Gynecology Clinic	Thomas K. Galvin
Gynecologists	Edward P. Smith J. J. Erwin Frank K. Morris Ernest S. Edlow H. L. Granoff Charles H. Doeller, Jr. William A. Dodd Harry McB. Beck William C. Duffy Joseph C. Sheehan William J. Rysanek Harry F. Kane Robert B. Tunney
Chief of Obstetrical Clinic	Edward P. Smith
Obstetricians	Harry F. Kane Charles H. Doeller, Jr. William A. Dodd Harry McB. Beck William C. Duffy Joseph C. Sheehan Robert B. Tunney William J. Rysanek, Jr.[1]
Esophagoscopist	Waitman F. Zinn
Associate Esophagoscopist	Fayne A. Kayser
Rhinologists and Laryngologists	Waitman F. Zinn Theodore A. Schwartz Benjamin H. Isaacs Ross C. Brooks

Ophthalmologists and Otologists	M. Raskin F. A. Pacienza Joseph V. Jeppi F. Edwin Knowles, Jr.
Supervisor of Proctology Clinic	Charles F. Blake
Proctologist	Simon H. Brager
Supervisor of Dental Clinic	J. D. Fusco
Consulting Dentist	Conrad L. Inman
Supervisor of Physiotherapy Clinic	Leon Hannan
Assistant Physiotherapist	Alice R. Hannan
Social Workers	Sister M. Helen Anna Shawbaker
Secretaries	Eva Applegarth Peggy O'Neill

MERCY HOSPITAL DISPENSARY REPORT

Year of 1946

Department	*New Cases*	*Old Cases*	*Total*
Allergy	15	7	22
Bronchoscopic	254	605	859
Cardiology	3	127	130
Dental	42	31	73
Dermatology	186	451	637
Diabetic	12	328	340
Gastro-Intestinal	12	88	100
Genito-Urinary	91	773	864
Gynecology	274	619	893
Medicine	422	2,188	2,610
Neurology	71	402	473
Ophthalmology	141	271	412
Orthopaedics	79	257	336
Pediatrics	236	782	1,018
Physiotherapy	109	1,531	1,640
Postnatal	32	128	160
Prenatal	356	1,791	2,147
Proctology	28	75	103
Rhinolaryngology	305	581	886
Surgery	730	4,653	5,383
Surgical Follow-Up	87	237	324
Well Baby Clinic	69	52	121
Totals	**3,554**	**15,977**	**19,531**

THE BALTIMORE CITY HOSPITALS

STAFF, 1947–1948

PARKER J. MCMILLIN, *Superintendent*

Consulting Chief Surgeon	ARTHUR M. SHIPLEY, M.D., D.Sc.
Surgeon-in-Chief	THOMAS B. AYCOCK, B.S., M.D.
Physician-in-Chief, Acting	C. HOLMES BOYD, M.D.
Physician-in-Chief, Radiology	STANLEY H. MACHT, B.S., M.D.
Physician-in-Chief, Tuberculosis Hospital	H. VERNON LANGELUTTIG, M.D.
Obstetrician-in-Chief	LOUIS H. DOUGLASS, M.D.
Pediatrician-in-Chief	HAROLD E. HARRISON, B.S., M.D.
Pathologist-in-Chief	C. GARDNER WARNER, A.B., M.D.
Dental Surgeon-in-Chief	LOWELL P. HENNEBERGER, D.D.S.
Consultant in Psychiatry	ESTHER L. RICHARDS, M.D.
Assistant Surgeon-in-Chief	OTTO C. BRANTIGAN, M.D.
Visiting Surgeons	JAMES C. OWINGS, M.D. HARRY C. HULL, JR., M.D. I. RIDGEWAY TRIMBLE, M.D. AMOS KOONTZ, M.D.
Consultant in Traumatic Surgery	CHARLES A. REIFSCHNEIDER, M.D.
Consultant in Peripheral Vascular Diseases	GEORGE H. YEAGER, B.S., M.D.
Consultants in Plastic Surgery	EDWARD A. KITLOWSKI, M.D. EDWARD M. HANRAHAN, JR., M.D.
Assistant Visiting Surgeons	HARRY C. BOWIE, M.D. THURSTON R. ADAMS, M.D.
Assistant Visiting Physician Tuberculosis	JOHN H. HIRSCHFELD, M.D.
Visiting Obstetrician	J. G. M. REESE, M.D.
Assistant Visiting Obstetricians	GEORGE W. ANDERSON, M.D. J. WILLIAM DORMAN, M.D. C. LOUIS GAREIS, M.D. D. FRANK KALTREIDER, M.D. JOHN E. SAVAGE, M.D. GILBERT J. VOSBURGH, M.D.
Assistant Pediatrician-in-Chief	WILSON E. HUGHES, M.D.
Visiting Pediatrician	HANS BIX, M.D.
Visiting Gynecologists	J. MASON HUNDLEY, JR., M.A., M.D. BEVERLY C. COMPTON, M.D. JOHN C. DUMLER, B.S., M.D. JOHN T. HIBBITTS, M.D.
Assistant Visiting Gynecologists	ERNEST I. CORNBROOKS, JR., M.D. WILLIAM K. DIEHL, M.D.

Assistant Chief Physician	Howard K. Rathbun, M.D.
Visiting Physicians	Louis A. M. Krause, M.D. Charles C. W. Judd, M.D.
Assistant Visiting Physicians	Dean F. Davies, M.D. James O. Davis, M.D.
Assistant Visiting Ophthalmologist	Charles E. Iliff, M.D.
Visiting Orthopaedic Surgeons	Allen Fiske Voshell, A.B., M.D. Henry F. Ullrich, M.D., D.Sc.
Assistant Visiting Orthopaedic Surgeon	Milton J. Wilder, M.D.
Visiting Laryngologists	John E. Bordley, M.D. Thomas R. O'Rourk, M.D. Fred T. Kyper, M.D.
Assistant Visiting Laryngologist	John H. Hirschfeld, M.D.
Visiting Urologist	W. Houston Toulson, M.Sc., M.D.
Assistant Visiting Urologists	Hugh Jewett, M.D. Harry S. Shelley, M.D. Howard B. Mays, M.D.
Visiting Neurological Surgeons	Charles Bagley, Jr., M.A., M.D. Richard G. Coblentz, M.A., M.D. James G. Arnold, Jr., M.D.
Assistant Visiting Neurological Surgeon	Raymond K. Thompson, M.D.
Visiting Neurologist	J. W. Magladery, M.D.
Assistant Visiting Neurologist	George G. Merrill, M.D.
Visiting Proctologist	Monte Edwards, M.D.
Visiting Oncologists	Grant E. Ward, A.B., M.D. Arthur G. Siwinski, M.D. Howard Jones, M.D.
Visiting Dermatologist	Franklin P. Grauer, M.D.
Visiting Dental Surgeon	H. Glenn Waring, D.D.S.
Assistant Visiting Dental Surgeon	Lawrence W. Bimestefer, D.D.S.
Visiting Laboratory Physician	Julius M. Waghelstein, M.D.
Physiologist	Nathan W. Shock, M.D.

THE JAMES LAWRENCE KERNAN HOSPITAL AND INDUSTRIAL SCHOOL OF MARYLAND FOR CRIPPLED CHILDREN

STAFF, 1947–1948

Surgeon-in-Chief and Medical Director	Allen Fiske Voshell, A.B., M.D.
Consultant in Orthopaedic Surgery and Roentgenology	Albertus Cotton, A.M., M.D.
Associate Orthopaedic Surgeons	Moses Gellman, B.S., M.D. Harry Rogers, M.D. Henry F. Ullrich, M.D. Winthrop M. Phelps, A.B., M.D.

Assistant Orthopaedic Surgeon	Milton J. Wilder, M.D.
Plastic Surgeon	Edward A. Kitlowski, A.B., M.D.
Aurist and Laryngologist	Benjamin S. Rich, A.B., M.D.
Dentist	M. E. Coberth, D.D.S.
Cardiologist	Helen M. Taussig, M.D.
Pediatrists	Mary L. Hayleck, M.D. W. Earl Weeks, M.D. Gibson J. Wells, M.D.
Consulting Surgeons	Arthur M. Shipley, M.D., Sc.D. Charles Reid Edwards, A.B., M.D. Thomas B. Aycock, B.S., M.D.
Consulting Aurists and Laryngologists	Franklin B. Anderson, M.D. Edward A. Looper, M.D., D.Oph.
Consulting Neurological Surgeon	Charles Bagley, Jr., M.A., M.D.
Consulting Plastic Surgeon	John Staige Davis, B.Sc., M.D.
Consulting Physician	Thomas R. Brown, A.B., M.D.
Consulting Dermatologists	Harry M. Robinson, Sr., M.D. Leon Ginsberg, M.D.
Consulting Neurologists	Irving J. Spear, M.D. R. V. Seliger, M.D.
Consulting Pediatrists	Benjamin Tappan, A.B., M.D. J. Edmund Bradley, M.D.
Consulting Dentist	Harry B. McCarthy, D.D.S.
Consulting Opthalmologists	Harry Friedenwald, A.B., M.D., D.H.L., D.Sc. William Tarun, M.D.
Consulting Pathologist	Hugh R. Spencer, M.D.
Consulting Roentgenologist	Henry J. Walton, M.D.
Resident Orthopaedic Surgeons	Samuel R. Terhune, M.D. George B. Barnes, M.D.
Superintendent	Miss Maud M. Gardner, R.N.
Dispensary and Social Service Nurse	Mrs. Evelyn Byrd Zapf, R.N.
Physical Therapists and X-ray Technician	Mrs. Joan Beck, P.T. Reg. Mrs. Georgiana Wisong
Occupational Therapist	Miss Muriel Zimmerman, O.T.
Instructor in Grammar School	Miss Bertha Sendelback

HISTORY OF THE SCHOOL OF MEDICINE

The present School of Medicine, with the title University of Maryland School of Medicine and College of Physicians and Surgeons, is the result of a consolidation and merger of the University of Maryland School of Medicine with the Baltimore Medical College (1913) and the College of Physicians and Surgeons of Baltimore (1915).

Through the merger with the Baltimore Medical College, an institution of thirty-two years' growth, the facilities of the School of Medicine were enlarged in faculty, equipment and hospital connection.

The College of Physicians and Surgeons was incorporated in 1872, and established on Hanover Street in a building afterward known as the *Maternité*, the first obstetrical hospital in Maryland. In 1878 union was effected with the Washington University School of Medicine, in existence since 1827, and the college was removed to Calvert and Saratoga Streets. Through the consolidation with the College of Physicians and Surgeons, medical control of the teaching beds in the Mercy Hospital was obtained.

The School of Medicine of the University of Maryland is one of the oldest foundations for medical education in America, ranking fifth in point of age among the medical colleges of the United States. It was organized in 1807 and chartered in 1808 under the name of the College of Medicine of Maryland, and its first class was graduated in 1810. In 1812 the College was empowered by the Legislature to annex three other colleges or faculties: Divinity, Law, and Arts and Sciences; and the four colleges thus united were "constituted an University by the name and under the title of the University of Maryland."

The original building of the Medical School at the N. E. corner of Lombard and Greene Streets was erected in 1812. It is the oldest structure in this country from which the degree of doctor of medicine has been granted annually since its erection. In this building were founded one of the first medical libraries and one of the first medical school libraries in the United States.

At this Medical School dissection was made a compulsory part of the curriculum, and independent chairs for the teaching of gynecology and pediatrics (1867), and of ophthalmology and otology (1873), were installed for the first time in America.

This School of Medicine was one of the first to provide for adequate clinical instruction by the erection of its own hospital in 1823. In this hospital intramural residency for senior students was established for the first time.

The School of Medicine has been co-educational since 1918.

BUILDINGS AND FACILITIES

The original medical building at the N. E. corner of Lombard and Greene Streets houses the office of the Dean, Room 101, the office of the Committee on Admissions, Room 102 and two lecture halls.

The Administration Building, to the east of the original building, contains the Baltimore offices of the Registrar and the Director of Admissions and two lecture halls.

The laboratory building at 31 South Greene Street is occupied by the departments of Pathology, Bacteriology and Biochemistry.

The Frank C. Bressler Research Laboratory provides the departments of Anatomy, Histology and Embryology, Pharmacology, Physiology and Clinical Pathology with facilities for teaching and research. It also houses the research laboratories of the clinical departments, animal quarters, a laboratory for teaching Operative Surgery, a lecture hall and the Bressler Memorial Room.

This building was erected in 1939–1940 at 29 South Greene Street opposite the University Hospital. It was built with funds left to the School of Medicine by the late Frank C. Bressler, an alumnus, supplemented by a grant from the Federal government. The structure, in the shape of an I, extends east from Greene Street, just north of the original building.

MEDICAL LIBRARY

Howard Rovelstad, A.B., M.A., B.S.L.S. . . . . *Acting Director of Libraries*

Ida Marian Robinson, A.B., B.S.L.S. . . . . *Librarian*
Mary Elizabeth Hicks, A.B., B.S.L.S. . . . . *Assistant Librarian*
Florence R. Kirk . . . . *Assistant Librarian*
Edith R. McIntosh, A.M., A.B.L.S. . . . . *Cataloguer*
Charlotte Jubb . . . . *Assistant to the Cataloguer*

The Medical Library of the University of Maryland, founded in 1813 by the purchase of the collection of Dr. John Crawford, now numbers 27,800 volumes and several thousand pamphlets and reprints. Over three hundred of the leading medical journals, both foreign and domestic, are received regularly. The library is housed in Davidge Hall, a comfortable and commodious building in close proximity to classrooms and laboratories, and is open daily for the use of members of the faculty, the student body and the profession generally. Libraries pertaining to particular phases of medicine are maintained by several departments of the medical school.

The library of the Medical and Chirurgical Faculty of Maryland and the Welch Medical Library are open to students of the medical school without charge. Other libraries of Baltimore are the Peabody Library and the Enoch Pratt Free Library.

DISPENSARY BUILDING

The old hospital building has been remodeled and is occupied by the Out-patient Department. Thus the students have been provided with a splendidly appointed group of clinics for their training in out-patient work. All departments of clinical training are represented in this remodeled building and all changes have been predicated on the teaching function for which this department is intended.

The office of the Medical School Physician is located in this building.

The Department of Art also occupies quarters here.

UNIVERSITY HOSPITAL

The University Hospital, which is the property of the University of Maryland, is the oldest institution for the care of the sick in the state of Maryland. It was opened in September 1823, under the name of the Baltimore Infirmary, and at that time consisted of but four wards, one of which was reserved for patients with diseases of the eye.

In 1933–1934 the new University Hospital was erected and patients were admitted to this building in November 1934. The new hospital is situated at the southwest corner of Redwood and Greene Streets, and is consequently opposite the medical school buildings. The students, therefore, are in close proximity and little time is lost in passing from the lecture halls and laboratories to the clinical facilities of the new building.

This new building, with its modern planning, makes a particularly attractive teaching hospital and is a very valuable addition to the clinical facilities of the medical school.

The new hospital has a capacity of 435 beds and 65 bassinets devoted to general medicine, surgery, obstetrics, pediatrics, and the various medical and surgical specialties.

The teaching zone extends from the second to the eighth floor and comprises wards for surgery, medicine, obstetrics, pediatrics, and a large clinical lecture hall. There are approximately 270 beds available for teaching.

The space of the whole north wing of the second floor is occupied by the department of roentgenology. The east wing houses clinical pathology and special laboratories for clinical microscopy, biochemistry, bacteriology, and an especially well appointed laboratory for students' training. The south wing provides space for electro-cardiographic and basal metabolism departments, with new and very attractive air-conditioned or oxygen therapy cubicles. The west wing contains the departments of rhinolaryngology and bronchoscopy, industrial surgery, ophthalmology, and male and female cystoscopy.

The third and fourth floors each provide two medical and two surgical wards. The fifth floor contains two wards for pediatrics, and on the sixth floor there are two wards for obstetrics. Each ward occupies the space of one wing of the hospital.

On the seventh floor is the general operating suite, the delivery suite, and the central supply station. The eighth floor is essentially a students' floor and affords a mezzanine over the operating and delivery suites, and a students' entrance to the clinical lecture hall.

In the basement there is a very well appointed pathological department with a large teaching autopsy room and its adjunct service of instruction of students in pathological anatomy.

The hospital receives a large number of accident patients because of its proximity to the largest manufacturing and shipping districts of the city.

The obstetrical service is particularly well arranged and provides accommodation for forty ward patients. This service, combined with an extensive home service, assures the student abundant obstetrical training.

During the year ending December 31, 1946, 2092 cases were delivered in the hospital and 675 cases in the outdoor department. Students in the graduating class observed at least thirty-five cases, each student being required to deliver at least ten patients in their homes.

The dispensaries associated with the University Hospital and the Mercy Hospital are organized upon a uniform plan in order that the teaching may be the same in each. Each dispensary has the following departments: medicine, surgery, pediatrics, ophthalmology, otology, genito-urinary, gynecology, gastroenterology, neurology, orthopaedics, proctology, dermatology, laryngology, rhinology, cardiology, tuberculosis, psychiatry, oral surgery and oncology.

All students in their junior year work each day during one-third of the year in the departments of medicine and surgery of the dispensaries. In their senior year, all students work one hour each day in the special departments.

MERCY HOSPITAL

The Sisters of Mercy first assumed charge of the Hospital at the corner of Calvert and Saratoga Streets, then owned by the Washington University, in 1874. By the merger of 1878 the Hospital came under the control of the College of Physicians and Surgeons, but the Sisters continued their work of ministering to the patients.

In a very few years it became apparent that the City Hospital, as it was then called, was much too small to accommodate the rapidly growing demands upon it. However, it was not until 1888 that the Sisters of Mercy, with the assistance of the Faculty of the College of Physicians and Surgeons, were able to lay the cornerstone of the present hospital. This building was completed and occupied late in 1889. Since then the growing demands for more space have compelled the erection of additions, until now there are accommodations for 348 patients.

In 1909 the name was changed from The Baltimore City Hospital to Mercy Hospital.

The clinical material in the free wards is under the exclusive control of the Faculty of the University of Maryland School of Medicine and College of Physicians and Surgeons.

THE BALTIMORE CITY HOSPITALS

The clinical facilities of the School of Medicine have been largely increased by the liberal decision of the Department of Public Welfare to allow the use of the wards of these hospitals for medical education. The autopsy material also is available for student instruction.

Members of the junior class make daily visits to these hospitals for clinical instruction in medicine, surgery, and the specialties.

The Baltimore City Hospitals consist of the following separate divisions:

The General Hospital, 400 beds, 90 bassinets.
The Hospital for Chronic Cases, 575 beds.
The Hospital for Tuberculosis, 280 beds.
Infirmary (Home for Aged) 700 beds.

THE JAMES LAWRENCE KERNAN HOSPITAL AND INDUSTRIAL SCHOOL OF MARYLAND FOR CRIPPLED CHILDREN

This institution is situated on an estate of 75 acres at Dickeyville. The site is within the northwestern city limits and of easy access to the city proper.

The location is ideal for the treatment of children, in that it affords all the advantages of sunshine and country air.

A hospital unit, complete in every respect, offers all modern facilities for the care of any orthopaedic condition in children.

The hospital is equipped with 100 beds—endowed, and city and state supported.

The orthopaedic dispensary at the University Hospital is maintained in closest affiliation and cares for the cases discharged from the Kernan Hospital. The physical therapy department is very well equipped with modern apparatus and trained personnel. Occupational therapy has been fully established and developed under trained technicians.

THE BALTIMORE EYE, EAR, AND THROAT HOSPITAL

This institution was first organized and operated in 1882 as an outgrowth of the Baltimore Eye and Ear Dispensary, which closed on June 14, 1882. The name then given to the new hospital was The Baltimore Eye and Ear Charity Hospital. It was located at the address now known as 625 W. Franklin St. The out-patient department was opened on September 18, 1882 and the hospital proper on November 1 of the same year. In 1898 a new building afforded 24 free beds and 8 private rooms; by 1907 the beds numbered 47; at present there are 60 beds, 29 of which are free. In 1922 the present hospital building at 1214 Eutaw Place was secured and in 1926 the dispensary was opened. In 1928 a clinical laboratory was installed. During 1943 the out-patient visits numbered 18,989.

Through the kindness of the Hospital Board and Staff, our junior students have access to the dispensary which they visit in small groups for instruction in ophthalmology.

REQUIREMENTS FOR ADMISSION

METHOD OF MAKING APPLICATION

Application forms may be filed one calendar year before the next incoming class. These forms may be secured from the Committee on Admissions, School of Medicine, University of Maryland, Baltimore-1, Maryland.

APPLICATION FOR ADMISSION TO THE FIRST YEAR

Application for admission is made by filing the required form and by having all pertinent data sent directly to the Committee on Admissions, in accordance with the instructions accompanying the application.

APPLICATION FOR ADMISSION TO ADVANCED STANDING

Students who have attended approved medical schools are eligible to file applications for admission to the second- and third-year classes. These applicants must be prepared to meet the current first-year entrance requirements in addition to presenting acceptable medical school credentials, and a medical school record based on courses which are quantitatively and qualitatively equivalent to similar courses in this school.

Application to advanced standing is made in accordance with the instructions accompanying the application form.

MINIMUM REQUIREMENTS FOR ADMISSION

The minimum requirements for admission to the School of Medicine are:

(a) Graduation from an approved secondary school, or the equivalent in entrance examinations, and

(b) Three academic years of acceptable college credit, exclusive of physical education and military sciences, earned in an approved college of arts and sciences. The quantity and quality of this course of study shall be not less than that required for recommendation by the institution where the college courses are being, or have been, studied.

The college curriculum shall include basic courses in

English
Biology
Inorganic Chemistry
Organic Chemistry
Physics
French or German

and such elective courses as will complete a balanced three-year schedule of study. The elective courses should be selected from the following three groups:

Humanities

- English (an advanced course in English composition should be taken, if possible)
- Scientific German or French (A reading knowledge of either language is desirable, although German is preferred)
- Philosophy

Natural Sciences

- Vertebrate Embryology
- Comparative Vertebrate Anatomy
- Quantitative Analysis
- Physical Chemistry
- Mathematics

Social Sciences

- Economics
- History
- Political Science
- Psychology (a basic course is desirable)
- Sociology, etc.

Careful attention should be given to the selection of elective courses in the natural sciences. Accordingly, it is suggested that the elective list given above be a guide in this connection and that the remainder of the college credits be accumulated from courses designed to promote a broad cultural development. Students should avoid the inclusion of college courses in subjects that occur in the medical curriculum, for example, histology, histological technique, human anatomy, bacteriology, physiology, neurology, physiological chemistry.

It is not intended that these suggestions be interpreted to restrict the education of students who exhibit an aptitude for the natural sciences or to limit the development of students who plan to follow research work in the field of medicine.

In accepting candidates for admission, preference will be given to those applicants who have acceptable scholastic records in secondary school and college, satisfactory scores in the Professional Aptitude Test, favorable letters of recommendation from their premedical committees, or from one instructor in each of the departments of biology, chemistry, and physics, and who in all other respects give every promise of becoming successful students and physicians of high standing.

Those candidates for admission who are unconditionally accepted will receive a certificate of matriculation from the office of the Dean.

COMBINED COURSE IN ARTS AND SCIENCES AND MEDICINE

A combined seven years' curriculum leading to the degrees of Bachelor of Science and Doctor of Medicine is offered by the University of Maryland. The first three years are taken in residence in the College of Arts and Sciences at College Park, and the last four years in the School of Medicine in Baltimore. (See University catalogue for details of quantitative and qualitative college course requirements.)

If a candidate for the combined degree completes the work of the first year in the School of Medicine with an average of "C" or better, and if he has absolved the quantitative and qualitative college requirements set up by the University, he is eligible to recommendation by the Dean of the School of Medicine that the degree of Bachelor of Science be conferred.

Because the general commencement at College Park usually takes place before the School of Medicine is prepared to release grades of the first-year class, this combined degree of Bachelor of Science is conferred at the commencement following the candidate's second year of residence in the School of Medicine.

STATE MEDICAL STUDENT QUALIFYING CERTIFICATES

Candidates for admission who live in or expect to practice medicine in Pennsylvania, New Jersey or New York, should apply to their respective state boards of education for medical student qualifying certificates (Pennsylvania and New Jersey) or approval of applications for medical student qualifying certificates (New York).

Those students who are accepted must file satisfactory State certificates in the office of the Committee on Admissions, School of Medicine, before registration. No exceptions will be made to this requirement.

Addresses of the State Certifying Offices

Director of Credentials Section, Pennsylvania Department of Public Instruction, Harrisburg, Pa.

Chief of the Bureau of Credentials, New Jersey Department of Public Instruction, Trenton, N. J.

Supervisor of Qualifying Certificates, The State Education Department, Examinations and Inspections Division, Albany, N. Y.

DEFINITION OF RESIDENCE STATUS OF STUDENTS*

Students who are minors are considered to be resident students if, at the time of their registration, the parents* have been residents of this State for at least one year.

Adult students are considered to be resident students if, at the time of their registration, they have been residents of this State for at least one year, provided such residence has not been acquired while attending any school or college in Maryland.

The status of the residence of a student is determined at the time of his first registration in the university and may not thereafter be changed by him unless, in the case of a minor, his parents* move to and become legal residents of this state by maintaining such residence for at least one full calendar year. However, the right of the student (minor) to change from a non-resident to a resident status must be established by him prior to registration for a semester in any academic year.

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* The term "parents" includes persons who, by reason of death or other unusual circumstances, have been legally constituted the guardians of or stand in loco parentis to such minor students.

CURRENT FEES

Matriculation fee (paid once)	$10.00
Tuition fee (each year)—Residents of Maryland	450.00
Tuition fee (each year)—Non-Residents	600.00
Laboratory fee (each year)	25.00
Student health service fee (each year)	20.00
Maintenance and service fee (each year)	10.00
Student activities fee (each year)	5.00
Graduation fee	15.00
Re-examination fee (each subject)	5.00
Transcript fee to graduates. First copy gratis, each copy thereafter	1.00

RULES FOR PAYMENT OF FEES

No fees are returnable.

Make all checks or money orders payable to the "University of Maryland".

When offering checks or money orders in payment of tuition and other fees, students are requested to have them drawn in the exact amount of such fees. Personal checks whose face value is in excess of the fees due will be accepted for collection only.

Acceptance.—Payment of the matriculation fee of $10.00 and of a deposit on tuition of $50.00 is required of accepted applicants before the expiration date specified in the offer of acceptance. This $60.00 deposit is not returnable and will be forfeited if the applicant fails to register, or it will be applied to the applicant's first semester's charges on registration.

Registration.—All students, after proper certification, are required to register at the business office, Gray Laboratory. (See calendar at front of this bulletin for dates for the payments of fees, and the note regarding late registration fee.)

One-half of the tuition fee, the laboratory fee, the student health fee, the maintenance and service fee and the student activities fee are payable on the date specified for registration for the first semester.

The remainder of the tuition fee shall be paid on the date designated for the payment of fees for the second semester. Fourth year students shall pay the graduation fee, in addition, at this time.

PENALTY FOR NON-PAYMENT OF FEES

If semester fees are not paid in full on the specified registration dates, a penalty of $5.00 will be added.

If a satisfactory settlement, or an agreement for settlement, is not made with the business office within ten days after a payment is due, the student automatically is debarred from attendance on classes and will forfeit the other privileges of the School of Medicine.

REEXAMINATION FEE

A student who is eligible to reexaminations must pay the business office $5.00 for each subject in which he is to be examined, and he must present the receipt to the faculty member giving the examination before he will be permitted to take the examination.

MAINTENANCE AND SERVICE FEE

This fee pays for the use of clothing lockers, and provides for library privileges and the maintenance of loan collections and of the student lounge and cafeteria. It also furnishes photographs for all departmental requests and for state board applications, pays room rent for the third year students when on outside obstetrics at Baltimore City Hospitals and furnishes the graduating students with invitations and tickets to the Pre-Commencement Exercises.

STUDENT ACTIVITIES FEE

This fee is used to maintain a recreational program for students of all classes in addition to supporting the activities of the Student Council.

PERSONAL EXPENSES

The following estimates of personal expenses for the academic year of eight months have been prepared by students, and are based upon actual experience. In addition to these the student must bear in mind the expenditure for a microscope.

	Low	*Average*	*Liberal*
Books	$50	$75	$100
College incidentals	20	20	20
Board, eight months	224	288	352
Room rent	144	184	216
Clothing and laundry	50	80	150
All other expenses	25	50	75
Total	$513	$697	$913

STUDENT HEALTH SERVICE

T. Nelson Carey, M.D. .... Director, Student Health Service

The Medical School has made provision for the systematic care of students according to the following plan:

1. *Preliminary Examination*—All new students will be examined during the first week of the semester. Notice of the date, time, and place of the examination will be announced to the classes and on the bulletin board. The passing of this physical examination is necessary before final acceptance of any student.

2. *Medical Attention*—Students in need of medical attention will be seen by the school physician, Dr. T. N. Carey, in his office at the medical school, at 9 A.M. daily, except Saturday and Sunday. In case of necessity, students will be seen at their homes.

3. *Hospitalization*—If it becomes necessary for any student to enter the hospital during the school year, the school has arranged for the payment of part or all of his hospital expenses, depending on the length of his stay and special expenses incurred. This applies only to students admitted through the school physician's office.

4. *Physical Defects*—Prospective students are advised to have any known physical defects corrected before entering school in order to prevent loss of time which later correction might incur.

5. *Eye Examination*—Each new matriculate is required to undergo an eye examination at the hands of an oculist (Doctor of Medicine) within three months before entering the School of Medicine. Long study hours bring out unsuspected

eye defects which cause much loss of time and inefficiency in study if not corrected until after school work is under way.

6. *Limitations*—It is not the function of this service to treat chronic conditions contracted by students before admission, nor to extend treatment to acute conditions arising in the period between academic years, unless the school physician recommends this service.

GENERAL RULES

The right is reserved to make changes in the curriculum, the requirements for graduation, the fees and in any of the regulations whenever the university authorities deem it expedient.

ADVANCEMENT AND GRADUATION

1. All students are required to take the spring examinations unless excused by the Dean.

2. When a student has failures in two completed major courses, he or she shall be dropped from the rolls of the Medical School.

3. No student will be permitted to pass to a higher class with conditions.

4. Should a student be required to repeat any year in the course, he must pay regular fees.

5. A student failing in final examinations for graduation at the end of the fourth year will be required to repeat the entire course of the fourth year and to take examinations in such other branches as may be required, should he again be permitted to enter the school as a candidate for graduation.

6. The general fitness of a candidate for graduation, as well as the results of his examinations, will be taken into consideration by the faculty.

EQUIPMENT

7. At the beginning of the first year, all students must be prepared to purchase microscopes of a satisfactory type equipped with a mechanical stage and a substage lamp.

A standard microscope of either Bausch & Lomb, Leitz, Spencer, or Zeiss make, fitted with the following attachments, will meet the requirements:

Oculars: 10 x and 5 x.
Triple nose piece with 16 mm., 4 mm., and 1.9 mm. 125 N.A. oil immersion lens.
Wide aperture stage with quick screw condenser.

All used microscopes are subject to inspection and approval before their use in the laboratory is permitted. The student is cautioned against the purchase of such an instrument before its official approval by the school.

8. Students in the second year class are required to provide stethoscopes.

9. Third- and fourth-year students are required to provide haemocytometers, opthalmoscopes and otoscopes.

STATE QUALIFYING CERTIFICATES

10. Candidates for admission who live in or expect to practice medicine in Pennsylvania, New Jersey or New York must file State certificates in the office of

the Committee on Admissions, School of Medicine, before registration. No exception will be made to this rule.

EYE EXAMINATION BEFORE ADMISSION

11. Each new matriculate in each class is required to present to the Committee on Admissions a certificate from an oculist, (a graduate in medicine) that the matriculate's eyes have been examined and are in condition, with or without glasses as the case may be, to endure the strain of close and intensive reading.

It is required that this examination be completed within three months prior to registration and that the certificate be mailed to the Committee on Admissions not later than one month before registration.

AWARDING OF COMBINED DEGREES

12. Students entering the School of Medicine on a three-year requirement basis from colleges which usually grant a degree on the successful completion of the first year of medicine, are restricted by the following regulations:

a—The candidate must present a certificate from his college or university that he has absolved the quantitative and qualitative premedical requirements for this degree.

b—The candidate must acquire an average of "C" or better for the work of his first year in the School of Medicine.

c—The Dean of the School of Medicine reserves the right to withhold his recommendation that a bachelor's degree be conferred at a commencement which occurs before the official release of first-year medical grades.

COST OF TRANSCRIPTS

13. Graduates will receive the first transcript of record without charge. Subsequent copies will cost one dollar each. Requests for transcripts must be filed with the Registrar's Office, University of Maryland, Lombard and Greene Streets, Baltimore-1, Maryland.

LIBRARY REGULATIONS

Loan Regulations

Loan periods have been worked out according to demand for and protection of different types of material.

Two-Week Loans: All books except those on reserve.

Three-Day Loans: All journals except the latest number (which does not circulate), and those on reserve.

Overnight Loans: Books and journals on reserve.
(4 p.m.–10 a.m.)

Special Rules or Books on Reserve:

Students whose names appear on the check-list for the Mercy Hospital section will be granted the necessary hours to return reserve books.

Overnight books may be reserved in advance only within the week in which they will be used. Books may be reserved on Saturday for the following Monday.

Overnight books may not be reserved two successive nights by the same person.

Advance reserves will be held until one hour before closing.

Fines

Fines are imposed not to acquire money, but to assure equal access to books.

Two-Week Loans: 5¢ per day.

Three-Day Loans: 5¢ per day.

Overnight Loans: 5¢ per hour.

Lost Books: List price of the book. (Lost books should be reported at once)

All books must be returned, lost books replaced or paid for, and fines paid before a student can finish the year in good standing.

In fairness to all concerned, these rules must be enforced without exception.

PRIZES

THE FACULTY PRIZE

The faculty will award the gold medal and five certificates of honor to those six of the first ten highest ranking candidates for graduation who, during the four academic years, have exhibited outstanding qualifications for the practice of medicine.

THE DR. A. BRADLEY GAITHER MEMORIAL PRIZE

A prize of $25.00 is given each year by Mrs. A. Bradley Gaither as a memorial to the late Dr. A. Bradley Gaither, to the student in the senior class doing the best work in genito-urinary surgery.

SCHOLARSHIPS

All scholarships are assigned for one academic year, unless specifically reawarded on consideration of an application.

Official application forms are obtainable at the Dean's office, where they should be filed four months before the ensuing academic year.

THE DR. SAMUEL LEON FRANK SCHOLARSHIP

(Value $100.00)

This scholarship was established by Mrs. Bertha Rayner Frank as a memorial to the late Dr. Samuel Leon Frank, an alumnus of this university.

It is awarded by the Trustees of the Endowment Fund of the University each year upon nomination by the Faculty Board "to a medical student of the University of Maryland, who in the judgment of said Council, is of good character and in need of pecuniary assistance to continue his medical course."

This scholarship is awarded to a second, third or fourth year student who has successfully completed one year's work in this school. No student may hold this scholarship for more than two years.

THE CHARLES M. HITCHCOCK SCHOLARSHIPS

(Value $100.00 each)

Two scholarships were established from a bequest to the School of Medicine by the late Charles M. Hitchcock, M.D., an alumnus of the university.

These scholarships are awarded annually by the Trustees of the Endowment Fund of the University, upon nomination by the Faculty Board, to students who have meritoriously completed the work of at least the first year of the course in medicine, and who present to the Board satisfactory evidence of a good moral character and of inability to continue the course without pecuniary assistance.

THE RANDOLPH WINSLOW SCHOLARSHIP

(Value $100.00)

This scholarship was established by the late Randolph Winslow, M.D., LL.D.

It is awarded annually by the Trustees of the Endowment Fund of the University, upon nomination by the Faculty Board, to a "needy student of the Senior, Junior, or Sophomore Class of the Medical School."

"He must have maintained an average grade of 85% in all his work up to the time of awarding the scholarship."

"He must be a person of good character and must satisfy the Faculty Board that he is worthy of and in need of assistance."

THE DR. LEO KARLINSKY MEMORIAL SCHOLARSHIP

(Value $125.00)

This scholarship was established by Mrs. Ray Mintz Karlinsky as a memorial to her husband, the late Dr. Leo Karlinsky, an alumnus of the university.

It is awarded annually by the Trustees of the Endowment Fund of the University, upon the nomination of the Faculty Board, to "a needy student of the Senior, Junior or Sophomore Class of the Medical School."

He must have maintained in all his work up to the time of awarding the scholarship a satisfactory grade of scholarship.

He must be a person of good character and must satisfy the Faculty Board that he is worthy of and in need of assistance.

THE UNIVERSITY SCHOLARSHIP

A scholarship which entitles the holder to exemption from payment of tuition fee for the year, is awarded annually by the Faculty Board to a student of the senior class in need of assistance who presents to the Faculty Board satisfactory evidence of good character and scholarship.

THE FREDERICA GEHRMANN SCHOLARSHIP

(Value $200.00)

(Not open to holders of Warfield and Cohen Scholarships)

This scholarship was established by the bequest of the late Mrs. Frederica Gehrmann and is awarded to a third-year student who at the end of the second year has passed the best practical examinations in physiology, pharmacology, pathology, bacteriology, immunology, serology, surgical anatomy and neuroanatomy.

THE CLARENCE AND GENEVRA WARFIELD SCHOLARSHIPS

(Value $300.00 each)

There are five scholarships established by the regents from the income of the fund bequeathed by the will of Dr. Clarence Warfield.

Terms and Conditions: These scholarships are available to students of any of the classes of the course in medicine. Preference is given to students from the counties of the state of Maryland which the Faculty Board may from time to time determine to be most in need of medical practitioners.

Any student receiving one of these scholarships must agree, after graduation and a year's internship, to undertake the practice of medicine, for a term of two years, in the county to which the student is accredited, or in a county selected by the Board. In the event that a student is not able to comply with the condition requiring him to practice in the county to which he is accredited by the Board, the money advanced by the regents shall be refunded by the student.

THE ISRAEL AND CECELIA E. COHEN SCHOLARSHIP

(Value $150.00)

This scholarship was established by the late Eleanor S. Cohen in memory of her parents, Israel and Cecelia E. Cohen. Terms and conditions: This scholarship will be available to students of any one of the classes of the course in medicine; preference is given to students of the counties in the state of Maryland which the Faculty Board may from time to time determine to be most in need of medical practitioners. Any student receiving one of these scholarships must, after graduation and a year's internship, agree to undertake the practice of medicine for a term of two years in the county to which the student is accredited, or in a county selected by the council. In the event that a student is not able to comply with the condition requiring him to practice in the county to which he is accredited by the Board, the money advanced by the regents shall be refunded.

THE DR. HORACE BRUCE HETRICK SCHOLARSHIP

(Value $125.00)

This scholarship was established by Dr. Horace Bruce Hetrick as a memorial to his sons, Bruce Hayward Hetrick and Augustus Christian Hetrick. It is to be awarded by the Faculty Board to a student of the senior class.

THE HENRY ROLANDO SCHOLARSHIP

(Value approximately $250.00)

The Henry Rolando Scholarship was established by the Board of Regents of the University of Maryland from a bequest to the Board by the late Anne H. Rolando for the use of the Faculty of Medicine.

This scholarship will be awarded each academic year on the recommendation of the Faculty Board to a "poor and deserving student."

THE READ SCHOLARSHIPS

The sum of $500.00 is now available to cover two (2) scholarships in the amount of $250.00 each for the scholastic year, beginning in 1945, as a donation from the Read Drug and Chemical Company of Baltimore, Maryland. Two students are to be selected by the Dean of the School in collaboration with the Scholarship and Loan Committee of the Medical School with the provision that the students selected shall be worthy, deserving students, residents of the State of Maryland.

LOAN FUNDS

W. K. KELLOGG FUND

This loan fund was established in the academic year 1942 with money granted by the W. K. Kellogg Foundation. The interest paid on the loans, together with the principal of the fund as repaid, will be used to found a rotating loan fund. Loans will be made on the basis of need, character and scholastic attainment.

FACULTY OF MEDICINE LOAN FUND

A Faculty of Medicine Loan Fund was established with money derived from the bequest of Dr. William R. Sanderson, Class 1882, and the gift of Dr. Albert Stein, Class 1907. Loans will be made on the basis of need, character, and scholastic ability.

THE JAY W. EATON LOAN FUND

This fund was established by the local chapter of the Nu Sigma Nu Fraternity in memory of Jay W. Eaton of the class of 1946.

Beginning in 1946 an interest-free loan of $100.00 will be made to some worthy member of the senior class, on recommendation of the Scholarship Committee of the School of Medicine. This loan is to be credited to the tuition fee of the appointed student and is to be repaid by the student within four years following his graduation.

THE SENIOR CLASS LOAN FUND

The senior class of 1945 originated this fund which will accumulate by subscription from among members of each senior class.

The conditions of the agreement provide that the dean of the School of Medicine award a loan of $100.00 to a needy member of the senior class on the recommendation of a self-perpetuating committee of two members of the faculty.

Loans from this fund are to be credited to the tuition fee of the appointed student and are to be repaid within five years from the date of graduation.

ORGANIZATION OF THE CURRICULUM

The curriculum is organized under thirteen departments.

1. Anatomy (including Histology and Embryology).

2. Physiology.
3. Bacteriology and Immunology.
4. Biological Chemistry.
5. Pharmacology and Materia Medica.
6. Pathology.
7. Medicine (including Medical Specialties).
8. Surgery (including Surgical Specialties).
9. Obstetrics.
10. Gynecology.
11. Ophthalmology.
12. Roentgenology.
13. Anaesthesiology.

The instruction is given in four academic years of graded work.

Several courses of study extend through two years or more, but in no case are the students of different years thrown together in the same course of teaching.

The first and second years are devoted largely to the study of the structures, functions and chemistry of the normal body. Laboratory work occupies most of the student's time during these two years.

Some introductory instruction in medicine and surgery is given in the second year. The third and fourth years are almost entirely clinical.

A special feature of instruction in the school is the attempt to bring together teacher and student in close personal relationship. In many courses of instruction the classes are divided into small groups and a large number of instructors insures attention to the requirements of each student.

In most courses the final examination as the sole test of proficiency has disappeared and the student's final grade is determined largely by partial examinations, recitations and assigned work carried on throughout the course.

DEPARTMENT OF GROSS ANATOMY

Eduard Uhlenhuth	Professor of Anatomy
Frank H. J. Figge	Professor of Experimental Anatomy
Vernon E. Krall	Associate Professor of Gross Anatomy
R. Dale Smith	Assistant Professor of Gross Anatomy
Karl F. Mech	Instructor in Gross Anatomy
Edmund B. Middleton	Weaver Fellow in Gross Anatomy[6]

Otto C. Brantigan	Professor of Surgical Anatomy
W. Wallace Walker	Assistant Professor of Surgical Anatomy
William B. Settle	Associate in Surgical Anatomy
Harry C. Bowie	Instructor in Surgical Anatomy
Herbert E. Reifschneider	Instructor in Surgical Anatomy
Ross Z. Pierpont	Assistant in Surgical Anatomy
Henry L. Rigdon	Assistant in Surgical Anatomy

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[6] June to September 9, 1947.

Gross Anatomy. *First Year.* First semester. Six lectures and conferences and seventeen laboratory hours a week during the first fourteen weeks; eleven lectures and conferences and twenty-seven laboratory hours a week during the following week.

This course comprises the dissection of the head, neck, chest, abdomen, pelvis and upper extremity; in addition, there is presented an extensive demonstration course of specially dissected preparations of the pelvis and perineum, and a separate lecture course on the peripheral nervous system. Members of the staff.

First Year. Second semester. One lecture and four laboratory hours a week during February and March, comprising the dissection of the lower extremity. Dr. Smith.

First semester	345 hours
Second semester	40 hours
Total	385 hours

Topographic and Surgical Anatomy. *Second Year.* The course is designed to bridge the gap between abstract anatomy and clinical anatomy as applied to the the study and practice of medicine and surgery. Students are required to dissect and to demonstrate all points, outlines, and regions on the cadaver. Underlying regions are dissected to bring out outlines and relations of structures.

Two lectures and two laboratory periods are given each week during the second semester. Drs. Brantigan, Walker, Settle, Bowie, H. E. Reifschneider, Pierpont and Rigdon.

Total hours: 96.

Advanced Anatomy (*elective course*). Selected problems in gross anatomy. This course is intended to offer to the advanced medical student and the postgraduate student an opportunity of extending the knowledge secured in the prescribed anatomy courses. Drs. Uhlenhuth and Brantigan.

In addition to the above course, facilities for special anatomical problems are offered to the more advanced student and physician.

Graduate Courses. Consult the catalogue of the Graduate School for descriptions of the graduate courses offered by members of the staff.

Postgraduate Courses. Consult the description of postgraduate courses in gross anatomy and surgical anatomy.

DEPARTMENT OF HISTOLOGY AND EMBRYOLOGY

Carl L. Davis	Professor of Anatomy
O. G. Harne	Associate Professor of Histology
John F. Lutz	Assistant Professor of Histology
[1]	Associate in Histology

First Year. The course in histology is divided equally between the study of the fundamental tissues and that of organs. Throughout the entire course the embryology of the part being studied precedes the study of the fully developed tissue. Thus embryology becomes a correlated part of the whole subject of microscopic anatomy and not an independent subject.

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[1] To be appointed

Each student is furnished a set of histological slides, previously prepared in our own laboratory, thus insuring a uniform and satisfactory quality of material for study and permitting the time of the student to be expended in the study of material rather than in the technic of its preparation. The object of the course is to present the evidence of function as shown by the structure of tissues and organs. Drs. Davis, Lutz and Harne.

An optional laboratory course is offered. This supplements the required course giving laboratory experience which can not be incorporated in the former. No added charge is made for the course.

Total hours: 150.

NEURO-ANATOMY

First Year. Neuro-anatomy embraces a study of the fundamental structure of the central nervous system as applied to its function. An abundance of material permits of individual dissection of the human brain. A series of appropriately stained sections of the human brain stem is furnished each student for the microscopic study of the internal structure of the nervous system. Drs. Davis, Lutz and Harne.

Total hours: 100.

Graduate Courses. Consult the catalogue of the Graduate School for descriptions of the graduate courses offered by members of the staff.

DEPARTMENT OF PHYSIOLOGY

William R. Amberson	Professor of Physiology
Dietrich Conrad Smith	Associate Professor of Physiology
Rubert S. Anderson	Assistant Professor of Physiology
Robert H. Oster	Professor of Physiology, School of Dentistry
Mary E. Chinn	Instructor in Physiology
June Linda Snyder	Assistant in Physiology

The work in physiology is given in two separate courses:

First Year. Second Semester. A course in neurophysiology is presented in two lectures a week, without laboratory work.

Second Year. First Semester. The remainder of the subject is presented in three lectures, one conference, and two laboratory periods a week.

The fundamental concepts of physiology are presented with special reference to mammalian problems.

Total hours: 224.

Graduate Courses. Consult the catalogue of the Graduate School for descriptions of the graduate courses offered by members of the staff.

DEPARTMENT OF BACTERIOLOGY AND IMMUNOLOGY

Frank W. Hachtel	Professor of Bacteriology
James G. McAlpine	Associate Professor of Bacteriology
H. Edmund Levin	Associate in Bacteriology
Henry F. Buettner	Instructor in Bacteriology

Second Year. First Semester. The principles of general bacteriology are taught by quiz, conference, and lecture.

Instruction given in the laboratory includes the methods of preparation of culture media, the study of pathogenic bacteria, and the bacteriological examination of water and milk. The bacteriological diagnosis of communicable diseases is also included.

Second Year. Second Semester. The principles of immunology are presented by means of quizzes, conferences and lectures.

The course includes a consideration of infection and immunity, the nature and action of the various antibodies, complement fixation and flocculation tests, hypersensitiveness, and the preparation of bacterial vaccines.

Experiments are carried out by the class in the laboratory. During the latter half of the semester the class is divided into sections.

Total hours: Bacteriology 120.
Immunology 72.

Graduate Courses. Consult the catalogue of the Graduate School for descriptions of the graduate courses offered by members of the staff.

DEPARTMENT OF BIOLOGICAL CHEMISTRY

H. Boyd Wylie	Professor and head of Biological Chemistry
Emil G. Schmidt	Professor of Biological Chemistry
Glenn S. Weiland	Assistant Professor of Biological Chemistry
Ann Virginia Brown	Instructor in Biological Chemistry

First Year. Second Semester. This course is designed to present the principles of biological chemistry and to indicate their applications to the clinical aspects of medicine. The phenomena of living matter and its chief ingredients, secretions and excretions are discussed in lectures and conferences and examined experimentally. Training is given in biochemical methods of investigation. Total hours: 208.

Graduate Courses. Consult the catalogue of the Graduate School for descriptions of the graduate courses offered by members of the staff.

DEPARTMENT OF PHARMACOLOGY

John C. Krantz, Jr.	Professor of Pharmacology
C. Jelleff Carr	Associate Professor of Pharmacology
Harry K. Iwamoto	Assistant Professor of Pharmacology
Joseph G. Bird	Assistant and Fellow in Pharmacology.
Ruth Musser	Instructor in Pharmacology
Stephen Krop	Lecturer in Pharmacology
Leonard Karel	Lecturer in Pharmacology
William G. Harne	Demonstrator in Pharmacology
Frederick K. Bell	U. S. Pharmacopoeia Fellow
Elaine Gaby	Emerson Fellow in Pharmacology
Mary J. Sauerwald	Emerson Fellow in Pharmacology
Edward B. Truitt, Jr.	Markle Fellow in Pharmacology

This course is designed to include those phases of pharmacology necessary for an intelligent use of drugs in the treatment of disease. The didactic instruction includes materia medica, pharmacy, prescription-writing, toxicology, posology, pharmacodynamics, and experimental therapeutics. The laboratory exercises parallel the course of lectures.

In addition, optional conference periods and lectures are available for students desiring further instruction or advice.

Total hours: 216.

Graduate Courses. Consult the catalogue of the Graduate School for descriptions of the graduate courses offered by members of the staff.

DEPARTMENT OF PATHOLOGY

Hugh R. Spencer	Professor of Pathology
Robert B. Wright	Associate Professor of Pathology
C. Gardner Warner	Associate Professor of Pathology
Walter C. Merkel	Associate Professor of Pathology
Albert E. Goldstein	Assistant Professor of Pathology
Dexter L. Reimann	Assistant Professor of Pathology
John A. Wagner	Assistant Professor of Pathology
Howard J. Maldeis	Associate in Pathology
Milton S. Sacks	Associate in Pathology
Benedict Skitarelic	Associate in Pathology
Conrad B. Acton	Instructor in Pathology
Howard B. Mays	Instructor in Pathology
Ephraim T. Lisansky	Instructor in Pathology
D. McClelland Dixon	Instructor in Pathology
William B. VandeGrift	Instructor in Pathology
Karl V. Mech	Instructor in Pathology
Seymour W. Rubin	Instructor in Pathology
William T. Raby	Assistant in Pathology
William J. Bryson	Assistant in Pathology
Isaac C. Wright	Assistant in Pathology

Courses of instruction in pathology are given during the second and third years. The courses are based on the previous study of normal structure and function and aim to outline the history of disease. The relationship between clinical symptoms and anatomical lesions is constantly stressed.

General Pathology. *Second Semester, Second Year.* This course includes the study of disturbances of the body fluids; disturbances of structure, nutrition and metabolism of cells; disturbances of fat, carbohydrate and protein metabolism; disturbances of pigment metabolism; inflammation and tumors.

Laboratory instruction is based on the study of prepared slides (loan collection) and corresponding gross material.

Applied Pathology, Including Gross Morbid Anatomy and Morbid Physiology. *Third Year.* The laboratory instruction in this course is carried out in small teaching museums where prepared specimens and material from autopsies with clinical histories and sections are available for study. For this work the class is divided into small groups. Clinical correlation is stressed.

Autopsies. *Third Year.* Students in small groups attend autopsies at the morgues of the University Hospital and the Baltimore City Hospitals.

Clinical-Pathological Conference. (*Fourth Year.*) These exercises are held in collaboration with the Department of Medicine. Selected cases are discussed and autopsy findings are presented.

Second year	184 hours
Third year	160 hours
Fourth year	30 hours
Total	374 hours

DEPARTMENT OF MEDICINE

Maurice C. Pincoffs	Professor of Medicine
G. Carroll Lockard	Professor of Clinical Medicine
Thomas P. Sprunt	Professor of Clinical Medicine
H. Raymond Peters	Professor of Clinical Medicine
Louis A. M. Krause	Professor of Clinical Medicine
T. Nelson Carey	Professor of Clinical Medicine
Paul W. Clough	Associate Professor of Medicine
Walter A. Baetjer	Associate Professor of Medicine
William S. Love, Jr.	Associate Professor of Medicine
Thomas C. Wolff	Associate Professor of Medicine
Howard M. Bubert	Associate Professor of Medicine
J. Sheldon Eastland	Associate Professor of Medicine
Milton S. Sacks	Associate Professor of Medicine
Lewis P. Gundry	Associate Professor of Medicine
William H. Smith	Associate Professor of Clinical Medicine
Howard J. Maldeis	Associate Professor of Legal Medicine
George McLean	Assistant Professor of Medicine
Wetherbee Fort	Assistant Professor of Medicine
Frank J. Geraghty	Assistant Professor of Medicine
H. Vernon Langeluttig	Assistant Professor of Medicine
Sol Smith	Assistant Professor of Medicine
Edward F. Cotter	Assistant Professor of Medicine
Theodore E. Woodward	Assistant Professor of Medicine
E. H. Tonolla	Associate in Medicine
Samuel Legum	Associate in Medicine
Robert A. Reiter	Associate in Medicine
W. Grafton Herspberger	Associate in Medicine
Meyer W. Jacobson	Associate in Medicine
Conrad B. Acton	Associate in Medicine
Irving Freeman	Associate in Medicine
Francis G. Dickey	Associate in Medicine
C. Edward Leach	Associate in Medicine
Lawrence M. Serra	Associate in Medicine
Marie A. Andersch	Associate in Medicine
Harry M. Robinson, Jr.	Associate in Medicine
William K. Waller	Associate in Medicine
Ephraim T. Lisansky	Associate in Medicine
Samuel T. R. Revell	Associate in Medicine

Arthur Karfgin	Associate in Medicine
M. Paul Byerly	Associate in Medicine
David Tenner	Instructor in Medicine
Philip D. Flynn	Instructor in Medicine
Edward S. Kallins	Instructor in Medicine
John A. Myers	Instructor in Medicine
Kurt Levy	Instructor in Medicine
William G. Helfrich	Instructor in Medicine
M. Paul Padget	Instructor in Medicine
Leon Ashman	Instructor in Medicine
S. Edwin Muller	Instructor in Medicine
Joseph E. Muse, Jr.	Instructor in Medicine
Daniel Wilfson, Jr.	Instructor in Medicine
William H. Kammer, Jr.	Instructor in Medicine
Leslie Harrell Pierce	Instructor in Medicine
Samuel J. Hankin	Instructor in Medicine
Frederick J. Vollmer	Instructor in Medicine
Louis J. Kroll	Instructor in Medicine
John R. Davis	Instructor in Medicine
John Z. Bowers	Instructor in Medicine
Wilfred H. Townshend	Instructor in Medicine
Alvin J. Hartz	Instructor in Medicine
Timothy Callahan	Instructor in Medicine
Morris Fine	Assistant in Medicine
Samuel Snyder	Assistant in Medicine
Joseph B. Gross	Assistant in Medicine
James R. Karns	Assistant in Medicine
L. Ann Hellen	Assistant in Medicine
Audry M. Funk	Assistant in Medicine
Rollin C. Hudson	Assistant in Medicine
Stephen J. Van Lill, 3rd	Assistant in Medicine
J. Douglas Lockard	Assistant in Medicine
Franklin E. Leslie	Assistant in Medicine
Sholom O. Waife	Assistant in Medicine
Thomas L. Worsley	Assistant in Medicine
John B. DeHoff	Assistant in Medicine
Adam Swiss	Assistant in Medicine
John W. Osborne	Assistant in Medicine

GENERAL OUTLINE

Second Year

Introduction to clinical medicine.

(a) Introductory physical diagnosis. (1 hour a week, first semester; 2 hours a week, second semester.)

(b) Medical clinics. (1 hour a week, second semester.)

Third Year

I. The methods of examination (13 hours a week). (a) History taking. (b) Physical diagnosis. (c) Clinical pathology.

These subjects are taught and practiced in the hospital out-patient department and in the clinical laboratory.

II. The principles of medicine (200 hours).
  (a) Lectures, clinics and demonstrations in general medicine, neurology, pediatrics psychiatry and preventive medicine.

III. The principles of therapeutics (15 hours). Lectures and demonstrations.

Fourth Year

The practice of medicine.

I. Clinical clerkship on the medical wards. (26 hours a week for ten weeks.)
  (a) Responsibility, under supervision, for the history, physical examination, laboratory examinations and progress notes of assigned cases.
  (b) Ward classes in general medicine, the medical specialties, and therapeutics.

II. Clinics in general medicine and the medical specialties. (6 hours a week.)

III. Dispensary work in the medical specialties.

IV. Clinical-pathological conferences (1 hour a week).

MEDICAL DISPENSARY WORK

The medical dispensaries of both the Mercy and the University Hospitals are utilized for teaching in the third year. Each student spends two hours daily for ten weeks in dispensary work. The work is done in groups of four to six students under an instructor. Systematic history-taking is especially stressed. Physical findings are demonstrated. The student becomes familiar with the commoner acute and chronic disease processes.

PHYSICAL DIAGNOSIS

Second Year. Didactic lectures and practical demonstrations in topographical anatomy and normal physical signs. Dr. McLean.

Third Year. The class is divided into small groups and each section receives instruction for two hours daily for ten weeks. This course is given at the Baltimore City Hospitals. The large clinical material there is utilized to give each student the opportunity to familiarize himself with the common types of bodily structure, with the normal variations in physical signs and with the physical signs of the chief pulmonary, circulatory and abdominal diseases. A course of lectures (1 hour a week) on physical diagnosis supplements the practical work in this subject. Drs. Wolff, Reiter, Ashman, Kroll, Legum and Wilfson.

THERAPEUTICS

Third Year. General therapeutics and materia medica are taken up and an effort is made to familiarize the student with the practical treatment of disease. The special therapy of the chief diseases is then reviewed.

Fourth Year. Special consideration is given to the practical application of therapeutic principles in bedside teaching and the chief therapeutic methods are demonstrated.

Students attend therapeutic ward rounds once a week throughout their medical trimester.

TROPICAL MEDICINE

Certain phases of tropical medicine are considered in the course on clinical pathology. In addition, a course of lectures and demonstrations is given to the entire fourth year class.

TUBERCULOSIS

During the third year in connection with the instruction in physical diagnosis a practical course is given at the Municipal Tuberculosis Hospital. Stress is laid upon the recognition of the physical signs of the disease, as well as upon its symptomatology and gross pathology.

CARDIOLOGY

In the third year a series of lectures and clinics correlated with pathological studies is given the entire class.

During the fourth year an elective course in cardiology is offered at the Mercy Hospital. The course occupies one and one-half hours weekly. Physical diagnosis, electocardiography and the therapeutic management of cardiac cases are stressed.

Elective out-patient work is available also to members of the fourth year class in the cardiac clinic of the University Hospital.

SYPHILIS

Third Year. During the third year the subject of syphilis is dealt with in the lecture course.

Fourth Year. An elective course in the therapeutic management of syphilis is offered in the dispensary.

CLINICAL PATHOLOGY

Milton S. Sacks	Associate Professor of Medicine and Head of Department of Clinical Pathology
Sol Smith	Assistant Professor of Medicine
Marie A. Andersch	Biochemist, University Hospital, Associate in Medicine
S. Edwin Muller	Instructor in Medicine
John A. Wagner	Assistant in Medicine
L. Ann Hellen	Assistant in Medicine
Audrey M. Funk	Assistant in Medicine

Third Year. First and second semesters. The course in Clinical Pathology is designed to train the student in the third year in the performance and interpretation of fundamental diagnostic laboratory procedures used in clinical medicine.

During the first semester the work is devoted to a thorough consideration of diseases of the hematopoietic system. In the second semester, in connection with laboratory work in urinalysis, gastric analysis, hepatic, pancreatic and renal functions, thorough discussion of underlying biochemical and physiological mechanisms is undertaken. During this semester examination of cerebrospinal fluid, transudates and exudates is also included. Elements of clinical parasitology round out the work in this semester.

Each student provides his own microscope and blood counting equipment. A completely equipped locker is assigned to every student.

Total Hours: 128.

Fourth Year. During the fourth year the student applies in the laboratories of the various affiliated hospitals the knowledge acquired during the preceding year. A completely equipped locker is assigned enabling him to work independently of the general laboratories. Instructors are available during certain hours to give necessary assistance and advice.

GASTRO-ENTEROLOGY

Theodore H. Morrison	Clinical Professor of Gastro-Enterology
Maurice Feldman	Assistant Professor of Gastro-Enterology
Zachariah Morgan	Assistant Professor of Gastro-Enterology
Samuel Morrison	Assistant Professor of Gastro-Enterology
Francis G. Dickey	Associate in Medicine
Z. Vance Hooper	Associate in Gastro-Enterology
Albert J. Shochat	Instructor in Gastro-Enterology
Alfred S. Lederman	Assistant in Gastro-Enterology

Third Year. A series of six lectures is given on the diseases of the digestive tract.

Fourth Year. Clinics and demonstrations are given to the class for one hour a week; dispensary instruction to small groups throughout the entire session. Practical instruction is given in the use of modern methods of study of the diseases of the gastro-intestinal tract.

PSYCHIATRY

Ross McC. Chapman	Professor of Psychiatry
Ralph P. Truitt	Professor of Clinical Psychiatry
H. W. Newell	Associate Professor of Psychiatry
Harry M. Murdock	Assistant Professor of Psychiatry
Philip S. Wagner	Assistant Professor of Psychiatry
Hans W. Loewald	Assistant Professor of Psychiatry
William W. Elgin	Associate in Psychiatry
Kathryn L. Schultz	Associate in Psychiatry
A. Russell Anderson	Instructor in Psychiatry
J. G. N. Cushing	Instructor in Psychiatry
Francis J. McLaughlin	Instructor in Psychiatry
Samuel Novey	Instructor in Psychiatry
Harold L. Vyner	Instructor in Psychiatry
Joseph H. Marshall	Instructor in Psychiatry
Richard H. Pembroke, Jr.	Instructor in Psychiatry

First Year. The 16 lecture hours will be devoted largely to a discussion of factors influencing the formation of character and the deviations in personality falling within the range of "normal." The usual endogenous and environmental experiences which provide critical periods during the life of an individual will be chronologically presented. Basic psychological concepts and psycho-dynamics will be reviewed. The methods of psychiatry with reference to the life history, mental status examination, and psychometric testing will be outlined and demonstrated.

Second Year. Fourteen 2-hour lecture demonstrations on psychopathology will introduce the student to personality deviations considered "abnormal." The mental status examination will be demonstrated in detail. The major and minor psychoses will be presented in terms of the psycho-dynamics of symptoms and reactoin types. The student will be expected, at the conclusion of the year, to be familiar with the psychopathology and clinical characteristics of the usual psychiatric problems.

Third Year. The 16 lecture hours will be devoted to further considerations of special psychopathology and the principles of psychotherapy. Specialized forms of treatment will be reviewed, but the main emphasis will be toward familiarizing the student with forms of therapy feasible in routine medical practice. During the 36 clinic hours the student will be supervised in history-taking, mental status and psychometric examination, and follow-up studies of patients.

Fourth Year. A series of 10 lecture demonstrations will serve to summarize previous instruction and to appraise the student's insight into psychopathology, the recognition of clinical syndromes, and their management. Characteristic reaction types will be demonstrated and discussed largely as concerns probable etiology and possible preventive measures. The relationship of mental illness to the major problems of social upheaval, economic, and other sources of insecurity will be considered.

PEDIATRICS

C. Loring Joslin	Professor of Pediatrics
Edgar B. Friedenwald	Professor of Clinical Pediatrics
Harold E. Harrison	Associate Professor of Pediatrics
Frederick B. Smith	Associate Clinical Professor of Pediatrics
J. Edmund Bradley	Associate Clinical Professor of Pediatrics
Albert Jaffe	Associate Clinical Professor of Pediatrics
A. H. Finkelstein	Associate Clinical Professor of Pediatrics
Samuel S. Glick	Assistant Professor of Pediatrics
Clewell Howell	Associate in Pediatrics
G. Bowers Mansdorfer	Associate in Pediatrics
Jerome Fineman	Associate in Pediatrics
Gibson J. Wells	Associate in Pediatrics
Arnold F. Lavenstein	Instructor in Pediatrics
Mary L. Hayleck	Instructor in Pediatrics
Israel P. Meranski	Instructor in Pediatrics
William Earl Weeks	Assistant in Pediatrics
J. Carlton Wich	Assistant in Pediatrics
A. Robert Marks	Assistant in Pediatrics
Elta C. Link	Assistant in Pediatrics
Walter Spurrier	Assistant in Pediatrics
Joseph M. Cordi	Assistant in Pediatrics

Third Year. The course is presented as follows:

Lectures on infant feeding and the fundamentals of diseases of infants and children. (15 hours.)

Lectures on contagious diseases in conjunction with the Department of Hygiene and Preventive Medicine. (14 hours.)

A special course in physical diagnosis is given at City Hospitals. (20 hours.)

Clinical conferences demonstrating diseases of the new-born. (6 hours.)

Fourth Year. An amphitheatre clinic is given at which patients are shown to demonstrate the features of the diseases discussed. (30 hours.)

Conferences and demonstrations are given in problems concerning diagnosis, care, treatment and clinical pathology of the diseases of infants and children. (30 hours.)

Students are assigned subjects for the preparation of theses.

Clinical clerkships are assigned on the pediatric wards, where experience is gained in taking histories, making physical examinations, doing routine laboratory work, and following up the patients' progress. This is under the supervision of the visiting staff. (140 hours.)

Instruction is given in the pediatric clinic of the out-patient department of the University Hospital. This consists of 1½ hours daily for five weeks—30 minutes each day being devoted to a clinical demonstration of some interesting case by a member of the staff; one hour daily to taking histories and making physical examinations under supervision of one of the staff instructors. (45 hours.)

Total hours: 300.

NEUROLOGY

Andrew C. Gillis	Professor of Neurology
Leon Freedom	Associate Professor of Neurology
Philip F. Lerner	Assistant Professor of Neurology
William L. Fearing	Associate in Neurology

Second Year. Fifteen one-hour lectures are given to correlate the anatomy and physiology of the nervous system with clinical neurology.

Third Year. Twenty hours of instruction are given to the whole class in neuropathology supplemented with pathological demonstrations. Sixteen lecture-demonstrations are given in which the major types of the diseases of the nervous system are discussed. A course is also given at the Baltimore City Hospitals, comprising eight periods of two hours each, in which the students in small groups carry out complete neurological examinations of selected cases which illustrate the chief neurological syndromes.

Fourth Year. A clinical conference one hour each week is given to the whole class at the University and Mercy Hospitals. All patients presented at these clinics are carefully examined. Complete written records are made by the students who demonstrate the patients before the class. The patients are usually assigned one or two weeks before they are presented, and each student in the class must study and present one or more patients during the year.

Ward Class Instruction. Nine hours of instruction are given to each student in small sections at the University and Mercy Hospitals. In these classes the students come in close personal contact with the patients in the wards under the supervision of the instructor.

Dispensary Instruction. Small sections are instructed in the dispensaries of the University and Mercy Hospitals five afternoons each week. In this way students are brought into contact with nervous diseases in their early and late manifestations.

HYGIENE AND PUBLIC HEALTH

Huntington Williams	Professor of Hygiene and Public Health
William H. F. Warthen	Associate Professor of Hygiene and Public Health
Ross Davies	Associate Professor of Hygiene and Public Health
Horace Hodes	Associate Professor of Hygiene and Public Health

Third Year. A one-hour lecture is given to the whole class each Tuesday during both semesters. Basic instruction is afforded in the clinical and public health aspects of the communicable diseases including syphilis and tuberculosis. The lectures are under the auspices of the Department of Medicine and are given by staff members of that department, including physicians representing pediatrics, hygiene and public health, and by staff members of the Baltimore City Health Department.

Fourth Year. Six one-hour lectures are given to the whole class scattered through the year on important public health procedures such as maternal and child hygiene, sewage disposal, vital statistics, interstate and international quarantine and research in public health. Elective work is also assigned at Sydenham Hospital, the one-hundred bed communicable disease hospital of the City Health Department, and at its Western Health District, 617 West Lombard Street, where the District Health Officer arranges for home visiting and the student prepares and presents a Home Survey Report.

The course deals with the fundamentals of public health and supplements the work in the third year. The major emphasis in both years is on the practice of preventive medicine and the relation of prevention to diagnosis and treatment, and on the civic and social implications of the medical services.

LEGAL MEDICINE

Howard J. Maldeis	Associate Professor of Legal Medicine Chief Medical Examiner, Maryland

Third Year. This course embraces a summary of some of the following: Proceedings in criminal and civil prosecution, medical evidence and testimony, identity and its general relations, rape, criminal abortions, signs of death, wounds in their medico-legal relations, natural and homicidal death, malpractice, insanity, and medico-legal autopsies, including poisoning.

Total hours: 4.

DEPARTMENT OF SURGERY

Arthur M. Shipley	Professor of Surgery
Walter D. Wise	Professor of Surgery
Charles Reid Edwards	Professor of Surgery

Elliott H. Hutchins	Professor of Surgery
Thomas B. Aycock	Professor of Clinical Surgery
F. L. Jennings	Professor of Clinical Surgery
D. J. Pessagno	Professor of Clinical Surgery
Monte Edwards	Clinical Professor of Surgery
Charles A. Reifschneider	Clinical Professor of Traumatic Surgery
Thomas R. Chambers	Associate Professor of Surgery
R. W. Locher	Associate Professor of Clinical Surgery
Edward S. Johnson	Associate Professor of Surgery
N. Clyde Marvel	Associate Professor of Surgery
Grant E. Ward	Associate Professor of Surgery
Cyrus F. Horine	Associate Professor of Surgery
Charles W. Maxson	Associate Professor of Surgery
George H. Yeager	Associate Professor of Surgery
C. W. Peake	Associate Professor of Surgery
Otto C. Brantigan	Associate Professor of Surgery
Harry C. Hull	Associate Professor of Surgery
H. F. Bongardt	Assistant Professor of Surgery
I. O. Ridgely	Assistant Professor of Surgery
James W. Nelson	Assistant Professor of Surgery
I. Ridgeway Trimble	Assistant Professor of Surgery
Richard T. Shackelford	Assistant Professor of Surgery
Simon H. Brager	Assistant Professor of Surgery
Thurston R. Adams	Assistant Professor of Surgery
W. Wallace Walker	Assistant Professor of Surgery
Raymond F. Helfrich	Associate in Surgery
E. Eugene Covington	Associate in Surgery
Luther E. Little	Associate in Surgery
William B. Settle	Associate in Surgery
Arthur G. Siwinski	Associate in Surgery
William F. Rienhoff, Jr.	Associate in Surgery
George Govatos	Associate in Surgery
Joseph V. Jerardi	Associate in Surgery
Henry L. Rigdon	Associate in Surgery
Joseph M. Miller	Lecturer in Surgery
E. M. Hanrahan	Instructor in Surgery
J. Duer Moores	Instructor in Surgery
Calvin Hyman	Instructor in Surgery
Clyde F. Karns	Instructor in Surgery
Daniel R. Robinson	Instructor in Surgery
Herbert E. Reifschneider	Instructor in Surgery
George H. Brouillet	Instructor in Surgery
Harry C. Bowie	Instructor in Surgery
William L. Garlick	Instructor in Surgery
Stuart G. Coughlan	Instructor in Surgery
Harold H. Burns	Instructor in Surgery
John F. Schaefer	Instructor in Surgery
Robert F. Healy	Instructor in Surgery
Robert C. Sheppard	Instructor in Surgery
Samuel E. Proctor	Instructor in Surgery

William R. Geraghty	Assistant in Surgery
Howard B. McElwain	Assistant in Surgery
A. V. Buchness	Assistant in Surgery
T. J. Touhey	Assistant in Surgery
Samuel H. Culver	Assistant in Surgery
Albert R. Wilkerson	Assistant in Surgery
L. T. Chance	Assistant in Surgery
W. Allen Deckert	Assistant in Surgery
F. Ford Loker	Assistant in Surgery
Patrick C. Phelan, Jr.	Assistant in Surgery
E. Roderick Shipley	Assistant in Surgery
Preston R. Cutler	Assistant in Surgery
James W. Hendrick	Assistant in Surgery
William C. Dunnigan	Assistant in Surgery
Howard L. Zupnik	Assistant in Surgery
Raymond M. Cunningham	Assistant in Surgery
Edwin H. Stewart, Jr.	Assistant in Surgery
Kenneth C. Sharrets	Assistant in Surgery
John W. Chambers	Assistant in Surgery

Instruction is given by means of lectures, laboratory work, recitations, dispensary work, bedside instruction, ward classes, and clinics. The work begins in the second year and continues throughout the third and fourth years.

The teaching is done in the anatomical laboratory, operative surgery laboratory, the dispensaries, wards, laboratories and operating rooms of the University and Mercy Hospitals, and in the wards and operating rooms of the Baltimore City Hospitals.

SECOND YEAR

Topographic and Surgical Anatomy. *Second semester.* The course is designed to bridge the gap between anatomy in the abstract and clinical anatomy as applied to the study and practice of medicine and surgery.

The teaching is done in the anatomical laboratory, and students are required to dissect and to demonstrate all points, outlines, and regions on the cadaver. Underlying regions are dissected to bring out outlines and relations of structures.

Two lectures and two laboratory periods per week. Drs. Brantigan, Walker, Settle, Bowie, H. E. Reifschneider and Rigdon.

Total hours: 96.

Principles of Surgery. *Second semester.* This course includes discussions of irritants, infection, repair of tissue, healing of tissue, relationship of bacteriology to surgery, modern chemotherapy in surgical diseases, ulcers, wounds, thrombophlebitis, phlebothrombosis, peripheral vascular diseases, thermal burns, injuries due to cold, surgical shock, diseases of the lymphatics, gangrene of the skin and extremities, aneurysms, hemorrhage, varicose veins, embolism, sinuses and fistulae, tetanus, anthrax and actinomycosis.

Lectures, two hours a week for one semester, are given to the whole class. Drs. Aycock and Bowie.

THIRD YEAR

General and Regional Surgery. Lectures, recitations and clinics on the principles of surgery, general surgery including fractures and dislocations are given three hours a week to the whole class. Drs. C. R. Edwards and Hull.

The class is divided into groups and receives instruction in history-taking and surgical pathology under the supervision of the chief of the pathologic department of the Baltimore City Hospitals. Instruction is also given in surgical diagnosis and in general surgery at the bedside and in the classroom at this institution by Drs. Aycock, C. A. Reifschneider, Brantigan and Adams. Two hours per week are given in orthopaedic surgery by Dr. Voshell, chief of the orthopaedic service of this institution.

Operative Surgery. Two courses in operative surgery are given under the supervision of Dr. Yeager assisted by Drs. Ullrich, Peake, Brager, Mays, Govatos, Jerardi, Karns, Robinson, Brouillet, Healy and Deckert. The class is divided into sections and each section is given practical and individual work under the supervision of the instructors.

Surgical Out-patient Department. Under supervision, the student takes the history, makes the physical examination, attempts the diagnosis and, as far as possible, carries out the treatment of ambulatory surgical patients in the University and Mercy Hospitals. Mercy Hospital—Dr. Raymond F. Helfrich assisted by the out-patient staff. University Hospital—Drs. Settle and Coughlan assisted by the out-patient staff.

FOURTH YEAR

Clinics. A weekly clinic is given at the Mercy and at the University Hospitals to one-half the class throughout the year. As far as possible this is a diagnostic clinic. Mercy Hospital—Dr. Wise. University Hospital—Dr. Shipley.

Surgical Pathology. At Mercy Hospital. Specimens from the operating room and museum are studied in the gross and microscopically in relation to the case history. 14 hours. Dr. Pessagno.

Surgery of the Chest. At Mercy Hospital. Operations and conferences. 14 hours. Drs. Rienhoff and Garlick.

Traumatic Surgery. This course deals with operative and post-operative treatment of accident cases and with instructions as to the relationship between the state, the employee, the employer, and the physician's duty to each. One hour a week to sections of the class throughout the year. Dr. C. A. Reifschneider.

Clinical Clerkship. This work includes the personal study of assigned hospital patients, under supervision of the staffs of the University and Mercy Hospitals, and embraces history-taking, and physical examination of patients, laboratory examinations, attendance at operations and observation of post-operative treatment.

Ward Classes. Ward-class instruction in small groups will consist of ward rounds, surgical diagnosis, treatment and the after-care of operative cases. Mercy Hospital—Drs. Wise, Hutchins, Blake, Pessagno, Nelson, Trimble, Brager and Jerardi. University Hospital—Drs. Shipley, C. Reid Edwards and Aycock.

DERMATOLOGY

Harry M. Robinson, Sr.	Professor of Dermatology
Francis A. Ellis	Assistant Professor of Dermatology
Harry M. Robinson, Jr.	Assistant Professor of Dermatology
Eugene S. Bereston	Associate in Dermatology
A. Albert Shapiro	Associate in Dermatology
Israel Zeligman	Associate Instructor in Dermatology
Lester N. Kolman	Instructor in Dermatology
Lucile J. Caldwell	Instructor in Dermatology
Mark B. Hollander	Instructor in Dermatology
Benjamin Highstein	Instructor in Dermatology
R. C. V. Robinson	Instructor in Dermatology
V. Harwood Link	Assistant in Dermatology

The third year class receives six lecture-demonstrations on the principles of dermatology by Dr. Robinson.

The senior course consists of demonstrations of the common skin diseases and venereal diseases given throughout the year by Dr. Robinson and staff. A weekly lecture-demonstration is given to the whole senior class by Dr. Robinson and Dr. Ellis.

Daily demonstrations and conferences are carried on by the out-patient staff in the dermatologic clinic involving both skin diseases and venereal diseases.

Third year	15 hours
Fourth Year	49 hours
Total	64 hours

ORTHOPAEDIC SURGERY

Allen Fiske Voshell	Professor of Orthopaedic Surgery
Compton Riely	Clinical Professor of Orthopaedic Surgery
Harry L. Rogers	Clinical Professor of Orthopaedic Surgery
Moses Gellman	Associate Professor of Orthopaedic Surgery
Henry F. Ullrich	Associate Professor of Orthopaedic Surgery
I. H. Maseritz	Associate in Orthopaedic Surgery
Milton J. Wilder	Instructor in Orthopaedic Surgery
Jason H. Gaskel	Instructor in Orthopaedic Surgery
John V. Hopkins	Assistant in Orthopaedic Surgery

Didactic, clinical, bedside and out-patient instruction is given in the fourth year at the University and Mercy Hospitals and Dispensaries, Kernan Hospital for Crippled Children at Dickeyville and Baltimore City Hospitals. Instruction is also given in the third year in small groups at the Baltimore City Hospitals.

Weekly lectures throughout the year present all phases of orthopaedic surgery except fractures; brief discussions and demonstrations of physical therapy are included.

Fourth year groups are given more intimate instruction biweekly at one of the above institutions; fracture cases are included here.

Third year	60 hours
Fourth year	90 hours
Total	150 hours

RHINOLOGY AND LARYNGOLOGY

Edward A. Looper	Professor of Rhinology and Laryngology
Waitman F. Zinn	Clinical Professor of Rhinology and Laryngology
Franklin B. Anderson	Associate Professor of Rhinology and Laryngology
Thomas R. O'Rourk	Associate Professor of Rhinology and Laryngology
Benjamin S. Rich	Associate Professor of Rhinology and Laryngology
W. Raymond McKenzie	Associate in Rhinology and Laryngology
Fayne A. Kayser	Associate in Rhinology and Laryngology
Theodore A. Schwartz	Associate in Rhinology and Laryngology
Frederick T. Kyper	Associate in Rhinology and Laryngology
Samuel L. Fox	Associate in Rhinology and Laryngology
Benjamin H. Isaacs	Instructor in Rhinology and Laryngology
Robert Z. Berry	Assistant in Rhinology and Laryngology
John H. Hirschfeld	Assistant in Rhinology and Laryngology

Third Year. Instruction to whole class is given in the common diseases of the nose and throat, attention being especially directed to infections of the accessory sinuses, the importance of focal infections in the etiology of general diseases and modern methods of diagnosis. Lectures illustrated by lantern slides are given one hour weekly for seven weeks by Dr. Looper.

Fourth Year. Dispensary instruction is given for one and one-half hours daily, to small sections at the University and the Mercy Hospitals. The student is afforded an opportunity to study, diagnose and treat patients under supervision. Ward classes and clinical demonstrations are given in periods of one and one-half hours weekly throughout the session in the University and the Mercy Hospitals.

The Looper Clinic for bronchoscopy and esophagoscopy, recently established in the University Hospital, affords unusual opportunities for students to study diseases of the larynx, bronchi and esophagus. The clinic is open to students daily from 2 to 4 P.M. under direction of Dr. Looper.

The Mercy Hospital clinic for bronchoscopy and esophagoscopy is under the direction of Dr. Zinn. In these two clinics the etiology, symptomatology, diagnosis and treatment of foreign bodies in the air and food passages, as well as bronchoscopy, are taught to students as an aid in the diagnosis and treatment of diseases of the lungs.

Third year	9 hours
Fourth year	53 hours
Total	62 hours

GENITO-URINARY SURGERY

W. HOUSTON TOULSON	Professor of Genito-Urinary Surgery
KENNETH D. LEGGE	Professor of Clinical Genito-Urinary Surgery
HOWARD B. MAYS	Assistant Professor of Genito-Urinary Surgery
FRANCIS W. GILLIS	Assistant Professor of Genito-Urinary Surgary
AUSTIN H. WOOD	Associate in Genito-Urinary Surgery
LYLE J. MILLAN	Associate in Genito-Urinary Surgery
L. K. FARGO	Associate in Genito-Urinary Surgery
JOHN F. HOGAN	Associate in Genito-Urinary Surgery
W. A. H. COUNCILL	Associate in Genito-Urinary Surgery
HUGH JEWETT	Assistant in Genito-Urinary Surgery
MORRIS A. FINE	Assistant in Genito-Urinary Surgery
JOHN S. HAINES	Assistant in Genito-Urinary Surgery

Third Year. This course is given for seven hours to the whole class. It consists of lectures and demonstrations, including the use of lantern slides and motion pictures. Dr. Toulson.

Fourth Year. The course in this year includes explanations and demonstrations of urethroscopy, cystoscopy, ureteral catheterization, renal function tests, urography, urine cultures and the various laboratory procedures. The teaching consists of clinics and ward rounds to small groups, and attendance by members of the senior class upon the out-patients in the dispensary. The student here is placed much on his own responsibility in arriving at a diagnosis. Members of the staff are in constant attendance for consultations. These dispensary classes are conducted at both the Mercy and University Hospitals where practically every variety of urogenital disease is seen and used for teaching purposes.

Third year	8 hours
Fourth year	64 hours
Total	72 hours

PROCTOLOGY

CHARLES F. BLAKE	Professor of Proctology
MONTE EDWARDS	Clinical Professor of Proctology
THURSTON R. ADAMS	Assistant Professor of Proctology
SIMON H. BRAGER	Associate in Proctology
DONALD B. HEBB	Assistant in Proctology
WILLIAM T. SUPIK	Assistant in Proctology
RAYMOND M. CUNNINGHAM	Assistant in Proctology

Third Year. Seven lectures are given to the whole class. This course is for instruction in the diseases of the colon, sigmoid flexure, rectum and anus, and covers the essential features of the anatomy and physiology of the large intestine as well as the various diseases to which it is subject. Dr. Monte Edwards.

Fourth Year. Ward and dispensary instruction is given in the University and Mercy Hospitals, where different phases of the various diseases are taught by direct observation and examination. The use of the proctoscope and sigmoidoscope in the examination of the rectum and sigmoid is made familiar to each student. Mercy Hospital—Drs. Blake and Brager. University Hospital—Drs. Monte Edwards and Adams.

Third year	7 hours
Fourth year	16 hours
Total	23 hours

OTOLOGY

Thomas R. O'Rourk	Clinical Professor of Otology
Franklin B. Anderson	Associate Professor of Otology
Benjamin S. Rich	Associate in Otology
William A. Parr	Instructor in Otology
Frederick T. Kyper	Instructor in Otology
Samuel L. Fox	Instructor in Otology

The course in otology is planned to give a practical knowledge of the anatomy and physiology of the ear, and its proximity and relationship to the brain and other vital structures. The inflammatory diseases, their etiology, diagnosis, treatment and complications are particularly stressed, with emphasis upon their relationship to the diseases of children, head-surgery and neurology.

Third Year. The whole class is given instruction by means of talks, anatomical specimens and lantern slides.

Fourth Year. Small sections of the class receive instruction and make personal examinations of patients under the direction of an instructor. The student is urged to make a routine examination of the ear in his ward work in general medicine and surgery.

Third year	12 hours
Fourth year	40 hours
Total	52 hours

NEUROLOGICAL SURGERY

Charles Bagley, Jr.	Professor of Neurological Surgery
Richard G. Coblentz	Clinical Professor of Neurological Surgery
James G. Arnold, Jr.	Associate Professor of Neurological Surgery
John A. Wagner	Assistant Professor of Pathology and Neuropathology
Raymond K. Thompson	Assistant in Neurological Surgery
Frank J. Otenasek	Assistant in Neurological Surgery
John W. Chambers	Assistant in Neurological Surgery
Robert M. N. Crosby	Fellow in Neurological Surgery

Third Year. The course covers instruction in diagnosis and treatment of surgical conditions of the brain, spinal cord and the peripheral nerves. Drs. Bagley, Coblentz, Arnold and Thompson.

Fourth Year. Weekly ward rounds and conferences are given at the University Hospital. Drs. Bagley, Coblentz, Arnold and Thompson.

Third year	12 hours
Fourth year	30 hours
Total	42 hours

ONCOLOGY

J. Mason Hundley, Jr.	Professor of Gynecology
Grant E. Ward	Associate Professor of Surgery
Beverley C. Compton	Associate in Gynecology
John C. Dumler	Associate in Gynecology
E. Eugene Covington	Associate in Surgery
Arthur G. Siwinski	Associate in Surgery
J. Duer Moores	Instructor in Surgery
Ernest I. Cornbrooks, Jr.	Instructor in Gynecology
William K. Diehl	Instructor in Gynecology
Everett S. Diggs	Assistant in Gynecology
Edwin H. Stewart, Jr.	National Cancer Institute Trainee
James W. Hendrick	National Cancer Institute Trainee

The purpose of the courses in Oncology is to give students training in the diagnosis and treatment of neoplastic diseases not obtained in other departments and at the same time to correlate this training with that received in surgery, medicine, roentgenology and other specialties.

Third Year: An out-patient clinic is held twice weekly, which affords an opportunity for instruction in small groups of students assigned in rotation from the general surgical and gynecological sections. The gynecological problems are under the supervision of Dr. Hundley and the general surgical conditions are under the direction of Dr. Ward.

In addition to dispensary work, five lectures in general oncology are given by Dr. Ward to the entire class at the end of the year. The increasing importance of the cancer problem throughout the State, Nation and civilized world is emphasized. An introduction to the history, physics and practical application of radium is given. The diagnosis and treatment, both surgical and radiological, of neoplasms of the head and neck, oral cavity, skin, breasts and hemopoietic system are also covered.

Fourth Year: Each ward class meets for one and one-half hours once a week for five weeks with Dr. Ward and staff for demonstration and discussion of patients with neoplastic diseases.

Dr. Hundley and staff give instructions in the diagnosis and treatment of cancer of the generative organs during the regular gynecological courses in addition to the above mentioned dispensary instruction.

Third year	8 hours
Fourth year	16 hours
Total	24 hours

DENTISTRY

[1]Brice M. Dorsey	Professor of Oral Surgery
Harry M. Robinson, Sr.	Professor of Dermatology
[1]Edward C. Dobbs	Professor of Pharmacology
Frederick C. Dye	Professor of Anaesthesiology

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[1] Faculty member, School of Dentistry.

[1]Vernon D. Kaufman	Associate Professor of Oral Surgery
[1]J. Herbert Wilkerson	Associate Professor of Oral Surgery
Grant E. Ward	Associate Professor of Oral Surgery
George McLean	Assistant Professor of Medicine
[1]Samuel H. Bryant	Instructor in Oral Diagnosis
[1]Eugene L. Pessagno	Instructor in Operative Dentistry
[1]Lewis C. Toomey	Instructor in Oral Surgery

This section has been reorganized for the teaching of both medical and dental students. There has been established a division in the out-patient department, and beds will be provided in the University Hospital, for the care of patients who will be available for the teaching of students from both schools.

Senior year: clinics weekly.

Ward instruction and group teaching are given. This includes diagnosis and treatment of diseases of the face, mouth and jaws.

INDUSTRIAL MEDICINE AND SURGERY

G. Carroll Lockard	Professor of Clinical Medicine
Charles A. Reifschneider	Clinical Professor of Traumatic Surgery
Thurston R. Adams	Assistant Professor of Surgery

This section is under the combined supervision of the medical and surgical departments. It is a cooperative effort by members of the medical school and hospital staff to afford means for clinical and laboratory study of the patient who has been subjected to traumatic or medical industrial hazard, so that adequate care may be instituted to promote his physical well-being. The facilities of the laboratories of the medical school and hospital are available as required.

Under direction of this department limited undergraduate instruction is given, especially in the methods of examination and of keeping records and in the general medico-legal principles as they affect the industrial employee, the employer, the general insurers, the physician and the hospital. There is also instruction on methods of making life insurance and other physical examinations, whether for employment or for health purposes. The wards of the University, Mercy and Baltimore City Hospitals provide for bed-side instruction.

Total hours: 48.

FIRST AID INSTRUCTION

Instruction consists of lectures combined with practical demonstrations and actual practice by students for one hour a week for ten weeks during the second semester of the Freshman Year.

PLASTIC SURGERY

Edward A. Kitlowski	Clinical Professor of Plastic Surgery
Clarence P. Scarborough	Assistant in Plastic Surgery
Edwin H. Stewart, Jr.	Assistant in Plastic Surgery
James W. Hendrick	Assistant in Plastic Surgery

This course is designed to acquaint students with the problems of reconstructive and plastic surgery. A subdivision in the dispensary has been established and beds for patients will be available for instruction in this course at the University and Baltimore City Hospitals and Kernan's Hospital for Crippled Children.

Third Year. Five lectures are given to the whole class. Dispensary instruction is provided on Mondays and Fridays.

Fourth Year. Ward rounds and operative demonstrations are held at the hospitals.

DEPARTMENT OF OBSTETRICS

Louis H. Douglass	Professor of Obstetrics
J. McFarland Bergland	Associate Professor of Obstetrics
Emil Novak	Associate Professor of Obstetrics
J. G. M. Reese	Associate Professor of Obstetrics
M. Alexander Novey	Assistant Professor of Obstetrics
Isadore A. Siegel	Assistant Professor of Obstetrics
John E. Savage	Assistant Professor of Obstetrics
Margaret B. Ballard	Associate in Obstetrics
Hugh B. McNally	Associate in Obstetrics
D. McClelland Dixon	Associate in Obstetrics
D. Frank Kaltreider	Associate in Obstetrics
Frank K. Morris	Instructor in Obstetrics
J. Huff Morrison	Instructor in Obstetrics
Kenneth B. Boyd	Assistant in Obstetrics
Thomas S. Bowyer	Assistant in Obstetrics
Maxwell L. Mazer	Assistant in Obstetrics
W. Kenneth Mansfield, Jr.	Assistant in Obstetrics
George H. Davis	Assistant in Obstetrics
Charles H. Doeller, Jr.	Assistant in Obstetrics
J. King B. E. Seegar, Jr.	Assistant in Obstetrics
Schuyler G. Kohn	Assistant in Obstetrics
John H. Morrison	Assistant in Obstetrics
Melvin D. Kappelman	Assistant in Obstetrics
William A. Mitchell	Assistant in Obstetrics
T. Edgie Russell, Jr.	Assistant in Obstetrics
L. Calvin Gareis	Assistant in Obstetrics

Third Year. The lectures and recitations consisting of three hours' teaching weekly are designed to cover the anatomy of the female generative tract and the bony pelvis, the physiology and development of the ovum and the physiology of pregnancy and labor. Following this the pathology of pregnancy, labor and the puerperium are taken up. Drs. Douglass, Novak, Reese, Siegel and Savage.

Each student spends time during his junior year at the Baltimore City Hospitals observing, assisting and finally delivering patients under strict supervision. Each student sees about twenty deliveries there, and does a considerable amount of the routine work.

The junior students are assigned as assistants to the seniors in the home delivery service and accompany them on deliveries.

Each student receives, in small groups, ten hours of instruction in palpation of patients and mensuration of the pelvis and demonstrations of the mechanism of labor. Drs. Siegel and McNally.

Fourth Year. At the weekly clinical conference, cases are presented and discussed and the student body is encouraged to offer opinions and to ask questions. There is no didactic teaching done, and an earnest effort is made to keep it, in every sense of the word, a conference. Dr. Douglass and associates.

The ward classes are held twice weekly for five weeks for each group. Various subjects are assigned and discussed, patients and their histories are presented. Drs. Reese, Novey, Savage and McNally.

Manikin instruction is given once a week. Drs. Dixon, Kaltreider and Doeller.

During the same five-week period, the students are sent into patients' homes to conduct deliveries under supervision of a senior member of the house staff and with the assistance of a graduate nurse. The student is held responsible for the complete conduct of each assigned case.

Each student spends thirty hours in the prenatal clinic, taking histories and examining patients under supervision.

Finally, the students are invited to attend the monthly meetings of The Committee on Maternal Mortality, where all maternal deaths occurring in Baltimore are openly discussed. *Hours*—Third year—148; Fourth year—102; total—250.

DEPARTMENT OF GYNECOLOGY

J. Mason Hundley, Jr.	Professor of Gynecology
Thomas K. Galvin	Clinical Professor of Gynecology
Leo Brady	Assistant Professor of Gynecology
Edward P. Smith	Assistant Professor of Gynecology
John T. Hibbitts	Associate in Gynecology
Kenneth B. Boyd	Associate in Gynecology
John C. Dumler	Associate in Gynecology
Beverley C. Compton	Associate in Gynecology
Houston Everett	Associate in Gynecology
J. J. Erwin	Associate in Gynecology
Frank K. Morris	Associate in Gynecology
Ernest I. Cornbrooks, Jr.	Instructor in Gynecology
Everett S. Diggs	Instructor in Gynecology
William K. Diehl	Instructor in Gynecology
Thomas S. Bowyer	Instructor in Gynecology
Ernest S. Edlow	Instructor in Gynecology
W. Allen Deckert	Instructor in Genecology
H. L. Granoff	Assistant in Gynecology
Helen I. Maginnis	Assistant in Gynecology
Charles H. Doeller, Jr.	Assistant in Gynecology
William A. Dodd	Assistant in Gynecology
John S. Haught	Assistant in Gynecology
Harry McB. Beck	Assistant in Gynecology
Charles B. Marek	Assistant in Gynecology
William C. Duffy	Assistant in Gynecology
Joseph C. Sheehan	Assistant in Gynecology
William J. Rysanek	Assistant in Gynecology
Harry F. Kane	Assistant in Gynecology
Robert B. Tunney	Assistant in Gynecology

Third Year. A course of thirty lectures and recitations is given to the whole class. In addition, a short course of lecture-demonstrations is given at the Baltimore City Hospitals, consisting of eight periods of one hour each, in which small groups of students are instructed in the fundamentals of gynecological diagnosis and examination.

Fourth Year. Operative clinics—lectures and demonstrations—are given six hours per week, for five weeks, to sections of the class.

Instruction in female urology is given. A small number of students may attend the cystoscopic dispensary which is held twice weekly.

The course in gynecology also includes instruction in the diagnosis and treatment of cancer of the generative organs. Small groups of students attend the oncological dispensary for additional work.

Third year	38 hours
Fourth year	74 hours
Total	112 hours

DEPARTMENT OF OPHTHALMOLOGY

Clyde A. Clapp	Professor of Ophthalmology
H. K. Fleck	Clinical Professor of Ophthalmology
Henry F. Graff	Clinical Professor of Ophthalmology
Jonas Friedenwald	Lecturer in Ophthalmic Pathology
Joseph I. Kemler	Associate in Ophthalmology
F. Edwin Knowles, Jr.	Associate in Ophthalmology
F. A. Holden	Instructor in Ophthalmology
Milton C. Lang	Instructor in Ophthalmology
A. Kremen	Instructor in Ophthalmology
Jerome Snyder	Assistant in Ophthalmology
Frederick M. Reese	Assistant in Ophthalmology

Third Year. Second semester. Dr. Graff reviews the anatomy and physiology of the eye and discusses the methods used in making the various examinations. Errors of refraction and their effect upon the general system are explained. Weekly section work, demonstrating the use of the ophthalmoscope, is carried on during the entire session.

Fourth Year. Clinics and demonstrations are given in diseases of the eye, weekly, for one year. Dr. Clapp.

This course consists of lectures upon the diseases of the eye, with particular reference to their diagnosis and relation to general medicine. Special lectures will be given upon vascular changes in the eye and upon the pathology of the eye. Some operations will be demonstrated by motion pictures.

Weekly ward classes are held at the University, The Baltimore Eye, Ear and Throat and Mercy Hospitals during which the eye grounds in the various medical and surgical conditions are demonstrated. Also daily demonstrations are given in the taking of histories and the diagnosis and treatment of the various conditions as seen in the dispensary. Drs. Kemler, Graff and Knowles.

Third year	20 hours
Fourth year	104 hours
Total	124 hours

DEPARTMENT OF ROENTGENOLOGY

Walter L. Kilby	Professor of Roentgenology
Charles N. Davidson	Associate Professor of Roentgenology
Asa D. Young	Assistant Professor of Roentgenology
Stanley H. Macht	Assistant Professor of Roentgenology
Donald J. Barnett	Associate in Roentgenology
Hugh D. Vazzana	Fellow in Roentgenology

During the academic year, small groups of the third and fourth year classes are given weekly instruction in the diagnostic and therapeutic uses of the Roentgen rays. An effort is made to familiarize the student with the indications for and the limitations of the Roentgen ray examinations. The history, physics and practical therapeutic application of Roentgen rays are given stressing the use of radiation as a weapon now available in a variety of disorders of the human body ranging from simple inflammations to malignant neoplastic conditions. Conferences are held with the various departments during the school year which are also open to members of the fourth year class.

Third year	8 hours
Fourth year	22 hours
Total	30 hours

DEPARTMENT OF ANAESTHESIOLOGY

Frederick C. Dye	Professor of Anaesthesiology
Alfred T. Nelson	Assistant Professor of Anaesthesiology

THIRD YEAR

Lectures are given on the general physiology and pharmacology of anesthesia, with consideration of the special physiology and pharmacology of each anaesthetic agent. The methods of induction and administration of anaesthesia are discussed. The factors influencing the selection of the anaesthetic are emphasized, and the preparation and care of the anaesthetized patient are carefully explained.

These lectures are correlated with practical demonstrations, supplemented by lantern slides and motion pictures, at the University Hospital.

FOURTH YEAR

Each senior student is required to spend two hours daily for two weeks observing and administering anaesthetics in the operating suite.

Third year	10 hours
Fourth year	24 hours
Total	34 hours

HISTORY OF MEDICINE

Louis A. M. Krause	Associate Professor of Medicine

Beginning with the spring of 1942 a group of lectures on the history of medicine has been presented on selected phases and trends of the development of medical knowledge and practice. It is planned to avoid duplication of subject matter for at least four years.

These lectures are offered primarily for our students, but a cordial invitation is extended to anyone who may wish to attend.

Announcement of the lectures will be made by mail and on the bulletin board of the School of Medicine.

ART AS APPLIED TO MEDICINE

Carl Dame Clarke	Associate Professor of Art as Applied to Medicine
Richard Dowell Grill	Instructor in Art as Applied to Medicine
Nancy Jane Ferneyhough	Fellow in Art as Applied to Medicine
Carl Christian Stein	Fellow in Art as Applied to Medicine

This department is maintained for the purpose of supplying pictorial and plastic illustrations for visual teaching in the classrooms of the medical school and for publication in scientific periodicals.

Special courses of instruction are given to qualified students.

POSTGRADUATE COURSES

Committee on Postgraduate Studies

Howard M. Bubert, *Chairman*
Dietrich C. Smith, *1st Vice-chairman*
L. A. M. Krause, *2nd Vice-chairman*
Milton S. Sacks, *Secretary*
John A. Wagner,
Otto C. Brantigan,
William K. Diehl,
John C. Krantz, Jr.,
Eduard Uhlenhuth,
Allen F. Voshell.

The Dean—*Ex Officio*

Immediately after the war, in response to a great number of requests, a review course in medicine, surgery, gynecology, obstetrics and pediatrics was given. This course lasted over a period of eight weeks. Because of the success achieved, the course was repeated, beginning on October 2, 1946 and running through November 27, 1946, and, whereas the participants were enthusiastic in their praise, the enrolment was small. Consequently, the Committee decided that the need for this particular type of course had been satisfied and the course was discontinued.

At the present time, the following courses are being given:

General Anatomy "A" This course is designed to prepare candidates for the anatomy examination of the American Board of General Surgery and Surgical Specialties. There is no hard and fast rule about either the content or duration of the course. Students may dissect a complete cadaver or any particular region in which they may be interested. Tuition arranged according to course content.

Surgical Anatomy "B" This course is designed to prepare candidates for the examination in Anatomy of the American Board of Surgery. This is a ninetyhour course, (2 days a week, 3 hours a day) given in conjunction with the regular sophomore medical course in surgical anatomy. Tuition: $150.00.

Pathology "A" This course offers basic training in general pathology in fulfillment of requirements as set up by the American Board of Pathology. Students will receive training in autopsy and surgical pathology. Minimum duration of the course is one year full time. Tuition: $300.00.

Pathology "B" This course is designed to prepare candidates for certification in surgery, surgical specialties and internal medicine. Individuals will receive training in autopsy and surgical pathology. Minimum duration is full time, six months. Tuition: $150.00.

Pathology "C" This course is designed to aid in meeting the requirements of the specialty boards in neurological sciences and covers basic studies in diseases of the central nervous system. Duration is six months, full time. Tuition: $200.00, plus $10.00 laboratory fee.

Gynecology and Obstetrics "A" This is a review for general practitioners. Duration is eighteen hours each of gynecology and obstetrics per week for twelve weeks. Tuition: $150.00.

Gynecology, Oncology and Female Urology "B" This is a review designed primarily for the general practitioner. Duration is ten weeks, full time. Tuition: $125.00.

Full descriptions of these courses are available.

Inquiries should be addressed to The Postgraduate Committee, University of Maryland School of Medicine, 29 S. Greene Street, Baltimore 1, Maryland.

FIRST YEAR SCHEDULE

FIRST SEMESTER, SEPTEMBER 18, 1947 TO JANUARY 24, 1948

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
9.00 to 12.00	*Histology and Embryology 11–12 lecture *Bressler 2*		Orientation (Sept. 24–Oct. 8) *Adm. 1*	*Histology and Embryology 11–12 lecture *Bressler 2*		Gross Anatomy A. H. 9–11
12.00 to 1.00	Lunch					
1.00 to 5.00	Gross Anatomy Lectures *A.H.* (1–2) Daily and Laboratories *Bressler 1* (2–5) Daily					

* Course ends January 16, 1948.

SECOND SEMESTER, JANUARY 26 TO JUNE 5, 1948

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
9.00 to 12.00	Laboratory — Biol. Chem. Sect. A	Laboratory — Biol. Chem. Sect. B	Laboratory — Biol. Chem. Sect. A	Laboratory — Biol. Chem. Sect. B	Neuro-Anatomy Laboratory	Neuro-Anatomy Laboratory
12.00 to 1.00	Lunch	Lunch	Lunch	Lunch	Lunch	
1.00 to 2.00	Biol. Chem. *Adm. 1*	Biol. Chem. *Adm. 1*	Biol. Chem. *Adm. 1*	Biol. Chem. *Adm. 1*	Biol. Chem. *Adm. 1*	
2.00 to 3.00	Neuro-Physiology *Bressler 2*	Neuro-Physiology *Bressler 2*	Biol. Chem. Conference *Adm. 1*	Gross Anatomy *Adm. 1* Feb. 12 to Apr. 8 — Neuro-Anatomy Apr. 15 Laboratory	Biol. Chem. Conference *Adm. 1*	
3.00 to 5.00		Gross Anatomy *Adm. 1* Feb. 10 to Apr. 6	Psychiatry (3–5) *C. H.*		(3–4) First Aid *C. H.* January 30 to April 9	

Locations of Lecture Halls and Laboratories:
Adm. 1—First Floor, Administration Building, Lombard and Greene Streets.
A. H.—Anatomical Hall—Upper Hall, N. E. Cor. Lombard and Greene Streets.
C. H.—Chemical Hall, Lower Hall, N. E. Cor. Lombard and Greene Streets.
Biological Chemistry Laboratory—Third Floor, 31 South Greene Street.
Bressler Research Laboratory—29 S. Greene Street.
Gross Anatomy—First Floor.
Histology and Embryology—Second Floor.
Neuro-anatomy—Second Floor.

Mid-Year Examinations—January 19–24, 1948
Final Examinations—May 24–29, 1948

SECOND YEAR SCHEDULE

FIRST SEMESTER, SEPTEMBER 18, 1947 TO JANUARY 24, 1948

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
8.30 9.30	Physiology *Bressler 2*	Physiology *Bressler 2*	Medicine *Bressler 2*	Physiology *Bressler 2*		Psychiatry (9–11) *Adm. 1*
9.30 to 10.30	Physiology Conference *Bressler 2*	Bacteriology *Adm. 1*		Pharmacology *Bressler 2*	Pharmacology *Bressler 2*	
10.30 to 12.30	†Bacteriology Laboratory				Neurological Diagnosis *C. H.*	
12.30	Lunch					
1.00 to 5.00	Pharm. Lect. (1–2) *Bressler 2* Pharmacology *B* Physiology *A*	Laboratory *A* Laboratory *B*		Pharmacology (1 to 4) *B* Physiology *A*	Laboratory *A* Laboratory *B*	

† Bacteriology Laboratory—Section work during the last month.

SECOND SEMESTER, JANUARY 26 TO JUNE 5, 1948

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
8.30 to 9.30	Surgery *Bressler 2*	Surgery *Bressler 2*	Surgical Anatomy *Bressler 2*	Medical Clinic *Amp.*	Physical Diagnosis *Adm. 1* 7 lectures	
9.30 to 10.30	Pharmacology *Bressler 2*	Pharmacology *Bressler 2*	Surgical Anatomy Laboratory *Bressler 1*		Pharmacology *Bressler 2*	
10.30 to 11.30	Pathology *C. H.*	Pathology *C. H.*		Pathology *C. H.*	Pathology *C. H.*	
11.30	Lunch					
12.00 to 2.00	Pathology Laboratory	Pathology Laboratory	Immunology Laboratory	Pathology Laboratory	Pathology Laboratory	
2.00 to 3.00	Surgical Anatomy *Adm. 1*	‖Immunology Laboratory		Pharmacology Laboratory *Sect. A*	Pharmacology Laboratory *Sect. B*	
3.00 to 5.00	Surgical Anatomy Laboratory *Bressler 1*		Optional period Pathology Immunology	Physical Diagnosis *Sect. B* (3.00–5.00) *U. H. D.*	Physical Diagnosis *Sect. A* (3.00–5.00) *U. H. D.*	

‖ Immunology Laboratory—Section work during last two months.

Locations of Lecture Halls and Laboratories:

Adm. 1—First floor, Administration Building, Lombard and Greene Streets.
C. H.—Chemical Hall, Lower Hall, Lombard and Greene Streets.
Amp.—Wilson Memorial Amphitheatre, New University Hospital, Greene and Redwood Streets, Eighth Floor.
U. H. D.—University Hospital Dispensary, Old Hospital Building.

Laboratories:
Physiology, Pharmacology, Surgical Anatomy—Bressler Building.
Bacteriology, Immunology, Pathology, Second Floor, 31 S. Greene Street.

Mid-Year Examinations—January 19–24, 1948
Final Examinations—May 24–29, 1948

THIRD YEAR SCHEDULE

SEPTEMBER 18, 1947 TO JUNE 5, 1948

SCHEDULE 1

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
8.30 to 9.20	(Whole Class) Obstetrics *C. H.* †Gynecology Mar. 22 to May 10	(Whole Class) Surgery *C. H.*	(Whole Class) Obstetrics *C. H.* †Gynecology Mar. 24 to May 12	(Whole Class) Surgery *C. H.*	(Whole Class) Pathology *C. H.*	(Whole Class) Surgery *C. H.* †Anaesthesiology Mar. 13 to May 15 *Amp.*
9.30 to 10.00	Transfer to Baltimore City Hospitals					
10.00 to 12.00	Physical Diagnosis, Pathology, Neurology and Pediatrics at B. C. H.					
12.00 to 1.00	Transfer and Lunch	Transfer and Lunch	Lunch	Transfer and Lunch	Lunch	
1.00 to 2.00	(Whole Class) Otology, Nose & Throat, Urology, Proctology, Plastic Surgery *C. H.*	(Whole Class) •Gynecology †Eye—10 wks. Jan. 27 to Apr. 6 †Oncology —5 wks. Apr. 13 to May 11 *C. H.*	Medical Clinic *B. C. H.*	(Whole Class) Clinical Pathology *Bressler 2*	Obstetrical Clinic *B. C. H.*	
2.00 to 4.00	(Whole Class) Pathology Laboratory *31*		Surgery — Pediatrics *B. C. H.*	(Whole Class) Clinical Pathology — Laboratory *Bressler 5*	Surgery — Pediatrics *B. C. H.*	
4.00 to 5.00	(Whole Class) •Physical Diagnosis, †Psychiatry, †Legal Medicine *C. H.*	(Whole Class) Hygiene and Public Health *C. H.*	Orthopaedics Roentgenology *B. C. H.*		Orthopaedics *B. C. H.*	

• First Semester. † Second Semester.

THIRD YEAR SCHEDULE
SEPTEMBER 18, 1947 TO JUNE 5, 1948

SCHEDULE 2

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
8.30 to 9.20	(Whole Class) Obstetrics *C. H.* †Gynecology Mar. 22 to May 10	(Whole Class) Surgery *C. H.*	(Whole Class) Obstetrics *C. H.* †Gynecology Mar. 24 to May 12	(Whole Class) Surgery *C. H.*	(Whole Class) Pathology *C. H.*	(Whole Class) Surgery *C. H.* Anaesthesiology Mar. 13 to May 15 *Amp.*
9.30 to 10.20	Pediatrics *C. H.*	*Medicine *C. H.*	*Medicine *C. H.*	*Therapeutics *C. H.*	*Medicine *C. H.*	Neurology *C. H.*
10.30 to 12.30	Operative Surgery—*Bressler 6* Medical and Surgical Dispensaries—(Univ. and Mercy Sections)					
12.30 to 1.00	Lunch					
1.00 to 2.00	Same as Schedule 1		Medical Clinic *Amp.*	Same as Schedule 1	Psychiatry Dermatology U. H. Disp.	
2.00 to 4.00			Ophthalmoscopy (5 weeks) *B. E. H.* — Obstetrics (5 weeks) Univ. Hosp. Disp. — Otology (5 wks.) *Univ. Hosp. 3-C*			
4.00 to 5.00					Obstetrics *Br. 2*	

* Pediatrics, May 4–14

The Junior Class will be divided into two sections—A and B. Each section reports to classes in keeping with the following schedule assignment, in which the letters represent the class sections and the numerals indicate the schedules to be followed for the periods shown.

Schedule Assignment

Periods	*Sections and Schedules*
September 18, 1947 to January 24, 1948	A-1, B-2
January 26 to May 15, 1948	B-1, A-2

Locations of Lecture Halls, etc.

A. H.—Anatomical Hall, Upper Hall, N. E. Cor. Lombard and Greene Streets.
Amp.—Wilson Memorial Amphitheatre, New University Hospital, Eighth Floor.
B. C. H.—Baltimore City Hosps., 4940 Eastern Ave.
B. E. H.—Baltimore Eye, Ear and Throat Hospital, 1214 Eutaw Place.
Bressler—Bressler Building, 29 S. Greene Street.
C. H.—Chemical Hall, Lower Hall, N. E. Cor. Lombard and Greene Streets.
Univ. Hosp.—New University Hospital, Greene and Redwood Streets.
U. H. Disp.—Old Hospital Building, S. W. Cor. Lombard and Greene Streets.
31—31 South Greene Street.
Clinical Pathology Laboratory—Fifth Floor, Bressler Building.
Pathology Laboratory—31 South Greene Street, Special Rooms, Basement.

Mid-Year Examinations—January 19-24, 1948
Final Examinations—May 17-29, 1948

FOURTH YEAR SCHEDULE

SEPTEMBER 18, 1947 TO JUNE 5, 1948

UNIVERSITY SECTION

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
9.00 to 11.00	Ward Classes — Medicine Surgery Obstetrics *Pediatrics	Ward Classes — Medicine Surgery Gynecology	Ward Classes — Medicine Surgery Obstetrics *Pediatrics	Ward Classes — Medicine Surgery Gynecology	Ward Classes — Medicine Surgery Obstetrics *Pediatrics	Ward Classes — Medicine Surgery Gynecology *Pediatrics
11.00 to 12.00	Clinical Clerkships	Ward Work	Clinical Pathological Conference *C. H.*	Surgical Clinic *Amp.*	Medical Clinic *Amp.*	Pediatric Clinic *Amp.*
12.00 to 2.00	Dispensary, Lunch	Dispensary, Lunch	Dispensary, Lunch	Dispensary, Lunch	Dispensary, Lunch	(12–1) Orthopaedic Clinic *Amp.*
2.15 to 3.15	Dermatology Clinic 1st time Full Class *Amp.* thereafter University Dispensary	Neurology Clinic *Amp.*	Eye and Ear Clinic (Full Class at Univ. Hosp.) *Amp.*	Obstetrical Clinic (Full Class at Univ. Hosp.) *Amp.*	Hygiene and Public Health (Full Class at Univ. Hosp.) *Amp.*	(1–2) *Dermatology *Amp.*
3.30 to 5.00	Ward Classes	Ward Classes	Ward Classes	Ward Classes	Ward Classes	
		Medicine		Therapeutics	Neurology	*Medical Section*
	See special schedule Medical School bulletin board	Proctology	Urology *Amp.*	Nose and Throat	Orthopaedic Surgery (*Kernan Hospital*)	*Surgical Section*
		Oncology (3.30–4.30) *Amp.*	Eye and Ear		Psychiatry *Amp.*	*Special Section*

(*)—Mercy special group, divided attendance at Univ. and Mercy.

University Sections on Medicine and Gynecology report at 8.30.

The Senior Class is divided into two sections, which report, one at Lombard and Greene Streets, the other at Calvert and Saratoga Streets for one semester each, then rotate.

Each section of the class is divided into three groups—Medical, Surgical, and Special. These groups will rotate on the following dates:

First Semester		*Second Semester*	
1st period	Sept. 18–Oct. 25	1st period	Jan. 26–Feb. 28
2nd period	Oct. 27–Nov. 29	2nd period	Mar. 1–Apr. 10
3rd period	Dec. 1–Jan. 17	3rd period	Apr. 12–May 8

C. H.—Chemical Hall—N. E. Cor. Lombard and Greene Streets.
Amp.—Wilson Memorial Amphitheatre—New University Hospital.
Room 34—Second floor, Calvert and Saratoga Streets.

Univ. Section Dispensary schedule posted in Old Hospital Bldg.
Mercy Section Dispensary schedule shown under Mercy Section.

Mid-Year Examinations—January 19–24, 1948

Final Examinations—May 10–15, 1948

FOURTH YEAR SCHEDULE

SEPTEMBER 18, 1947 TO JUNE 5, 1948
MERCY SECTION

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	
9.00 to 10.00	Clinical Clerkships	Surgical Pathology or Chest Surgery Room 34	Clinical Pathological Conference Room 34	Surgical Clinic Room 34	Medical Clinic Room 34	Pediatric Clinic Room 34	
10.00 to 12.00	Medical Ward Work	Medical Ward Work	Medical Ward Work	Medical Ward Work	Medical Ward Work	Medical Ward Work	*Medical Section*
	Medical Dispensary Group A	Medical Dispensary Group B	Medical Dispensary Group A	Medical Dispensary Group B	Medical Dispensary Group A	Medical Dispensary Group B	
	Surgical Ward Work	Surgical Ward Work	Surgical Ward Work	Surgical Ward Work	Surgical Ward Work	Surgical Ward Work	*Surgical Section*
	*Pediatrics	Gynecology — *Pediatrics	*Pediatrics	Gynecology — *Pediatrics	*Pediatrics	*Pediatrics	*Special Section*
12.00 to 2.00	Lunch, Dispensary	Lunch, Dispensary	Lunch, Dispensary	Lunch, Dispensary	Lunch, Dispensary	(12–1) Orthopaedic Clinic Room 34	
	Neurology	G.I.	Neurology	G.I.	Neurology		Medical A
	Orthopaedic	Dermatology	Orthopaedic	Dermatology	Orthopaedic	(1–2) Dermatology	Medical B
	G.U.	G.U.	Follow up Surgical Clinic	G.U.	G.U.		Surgical A
	N. & T.	Proctology	N. & T.	Proctology	N. & T.		Surgical B
	Pediatrics (U.H.)		Pediatrics (U.H.)	Pediatrics (U.H.)	Pediatrics (U.H.)		Special A
	Pediatrics (U.H.)		Pediatrics (U.H.)	Pediatrics (U.H.)	Pediatrics (U.H.)		Special B
2.15 to 3.15	Dermatology Clinic 1st time Full Class *Amp.* thereafter University Dispensary	Neurology Clinic Room 34	Eye and Ear Clinic (Full Class at Univ. Hosp.) *Amp.*	Obstetrical Clinic (Full Class at Univ. Hosp.) *Amp.*	Hygiene and Public Health (Full Class at Univ. Hosp.) *Amp.*		
3.30 to 5.00	See Special Schedule	Medicine	G.I.	Medicine	Neurology		Medical Section
		Orthopaedics	N. & T.	Proctology	Roentgenology		Surgical Section
		Pediatrics	Public Health	Pediatrics (U.H.)	Psychiatry (U.H.)		Special Section

* Mercy special group, divided attendence at Univ. and Mercy.

University Sections on Medicine and Gynecology report at 8.30.

The Senior Class is divided into two sections, which report, one at Lombard and Greene Streets, the other at Calvert and Saratoga Streets for one semester each, then rotate.

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1st period	Sept. 18–Oct. 25	1st period	Jan. 26–Feb. 28
2nd period	Oct. 27–Nov. 29	2nd period	Mar. 1–Apr. 10
3rd period	Dec. 1–Jan. 17	3rd period	Apr. 12–May 8

C.H.—Chemical Hall—N. E. Cor. Lombard and Greene Streets.
Amp.—Wilson Memorial Amphitheatre—New University Hospital
Room 34—Second floor, Calvert and Saratoga Streets.

Univ. Section Dispensary schedule posted in Old Hospital Bldg.

Mercy Section—Dispensary schedule at Mercy Hospital shown under Mercy Section, herewith.

Mid-Year Examinations—January 19–24, 1948

Final Examinations—May 10–15, 1948

UNIVERSITY OF MARYLAND SCHOOL OF MEDICINE AND COLLEGE OF PHYSICIANS AND SURGEONS

GRADUATES, JUNE 7, 1947

Abell, Harry Davis.........Kentucky
Allen, Gladys Martha, *B.S.*..Maryland
Anthony, James Elmer, Jr., *B.S.* Maryland
Beatty, John Orville, *B.S.*.....Nevada
Benson, John Fisher, *B.S.*....Maryland
Berggreen, Raymond George, *A.B.* Minnesota
Bindeman, William Wylie, *B.S.* North Carolina
Blair, James Seaborn, Jr. North Carolina
Blevins, Joseph Wendell, *B.S.* Pennsylvania
Brickner, John George, III, *B.S.* Maryland
Bunch, Littleton Jay, *A.B.* North Carolina
Bunn, David Glenn......North Carolina
Carouge, Gilbert Morris.....Maryland
Chase, Henry Vincent, *Ph.B.*.Maryland
Coffey, Michael Joseph, *B.S.* New Jersey
Cohen, Bernard Stanley.....Maryland
Cohen, Irvin, *A.B.*..........Maryland
Cooper, Harry Douglas, *B.S.*.Maryland
Cooper, William Benjamin, Jr., *B.S.* Maryland
Corzine, William Jenkins, Jr. North Carolina
Coultas, Elizabeth Louise, *A.B.* New Jersey
Cushner, Irvin Morris.......Maryland
Dorman, Parker Sturgis, *A.B.* District of Columbia
Duvall, Robert Caywood, Jr..Maryland
Eppes, Williford, *B.S.*........Maryland
Evans, John Ebenezer, Jr., *B.S.* North Carolina
Faraino, Frank Anthony, *B.S.* Maryland
Fink, Joel Charles, *B.S.*......Alabama
Fisher, Donald Ernst........Maryland
Fisher, George William, *B.S.*.Maryland
Fitzgerald, Robert Greeson, Jr., *B.S. in Medicine*.......North Carolina
Forrer, Gordon Randolph, *B.S.* Maryland
Friedman, Asher Authur, *A.B.*.Virginia
Gakenheimer, William Alfred Maryland
Gardner, Robert Kline......Maryland
Geddes, David Kofoid, *B.S.*..California
Gerlach, Jane Knowles, *A.B.*.Maryland
Gillson, Edward Michael, *A.B.* New Jersey
Gold, Benjamin Miller..North Carolina
Grau, Edward Gordon, *B.S.*..Maryland
Grossman, Louis Ward, Jr..Pennsylvania
Haase, Frederick Robert....Maryland
Hahn, Robert Roland........Maryland
Hall, Howard Essex.........Maryland
Hearne, Calvin Beck............Ohio
Hightower, John Allen........Florida
Hogan, John Francis, Jr......Maryland
Hooks, Richard Eugene..North Carolina
Houghton, James Francis....New Jersey
Hunter, Robert Carl, *B.S.*...Maryland
Imbrie, David Ellwood, *A.B.* Pennsylvania
Jernigan, Lane McDermott..Maryland
Judd, Allyn Francis, *A.B.*.Massachusetts
Leung, Bernard, *B.S.*.........New York
Levin, Norman...............Maryland
LiPira, Joseph Francis, *B.S.*..Maryland
Machata, Francis Karl, *Ph.B.* New York
Mannino, Peter Paul........Maryland
Mansberger, Arlie Roland, Jr. West Virginia
Mattern, Carl Frederick Theodore Maryland
Mattern, Elizabeth Anne Dentry, *A.B.*......................Maryland
Mitchell, Wallace Herttell, *B.S.* Florida
Morrow, John Gordon, Jr.....Georgia
Nachlas, Nathan Edward....Maryland
Pertz, Elden Hobart, *B.S.*.West Virginia
Phillips, David Lee, *A.B.*.....Maryland
Piggott, John Burr, Jr., *B.S.* District of Columbia
Post, William Reyman, *A.B.* Mississippi
Powell, Jack Hanson, Jr., *A.B.* Georgia
Rasch, George Chesley, Jr., *B.S.* Maryland
Ribet, James Ernest....North Carolina
Rogers, Joseph Megeath......Virginia
Rosser, John Hays, *B.S.*.......Maryland
Sachs, Sidney Herman........Maryland
Salvati, Eugene Philip, *A.B.* West Virginia
Schapiro, Joseph Solomon....Maryland
Schnitzker, William Frederic Maryland
Scott, Harry Baty.............Virginia
Senning, Frederick Stephen..Maryland
Shaw, John Hill, *B.S.*.........Virginia
Shear, Joseph................Maryland
Shulman, Melvin ....Maryland
Silber, Earle, *A.B.*..........Maryland
Smith, John Richard, Jr., *B.S.*.Maryland

Spino, Pascal Daniel.....Pennsylvania
Stenstrom, William Harold, *B. S.* Washington
Sussman, Harold, *A.B.*........Maryland
Swink, Robert Landrum.......Florida
Trench, James McKechan, *B.S.* Pennsylvania
Valderas, Jose Gervasio...Puerto Rico
Venable, Sidney Johnson, Jr., *B.S.* Maryland
Waltz, Robert Claude, *B.S.*......Ohio
White, John Phillips, III.....Maryland
Williams, James Delbert....California
Williams, John Elbert......California
Winokur, George, *A.B.*........Maryland
Zinberg, Norman Earl, *B.S.*..Maryland

HONORS

University Prize Gold Medal

Milton Reisch

Certificates of Honor

Williford Eppes
William Alfred Gakenheimer
John George Brickner, III
Bernard Stanley Cohen
Harold Sussman

INTERNSHIPS—GRADUATES OF JUNE 7, 1948

July 1, 1947–June 30, 1948

Abell, Harry Davis, Jr.	St. Louis City Hospital, St. Louis, Mo.
Allen, Gladys Martha	Hospital of the Woman's Medical College, Philadelphia, Pa.
Anthony, James Elmer, Jr.	Jefferson Hospital, Roanoke, Va.
Beatty, John Orville	Indiana University Medical Center, Indianapolis, Ind.
[2]Benson, John Fisher	University Hospital, Baltimore, Md.
Berggreen, Raymond George	Jersey City Hospital, Jersey City, N. J.
Bindeman, William Wylie	West Baltimore General Hospital, Baltimore, Md.
Blair, James Seaborn, Jr.	University of Virginia Hospital, Charlottesville, Va.
Blevins, Joseph Wendell	Hamot Hospital, Erie, Pa.
Brickner, John George, III	Mercy Hospital, Baltimore, Md.
Bunch, Littleton Jay	Colorado General Hospital, Denver, Col.
Bunn, David Glenn	Medical College of Virginia, Hospital Division, Richmond, Va.
Carouge, Gilbert Morris	South Baltimore General Hospital, Baltimore, Md.
Chase, Henry Vincent	Mercy Hospital, Baltimore, Md.
Coffey, Michael Joseph	Fitkin Memorial Hospital, Neptune, N. J.
Cohen, Bernard Stanley	Sinai Hospital, Baltimore, Md.
Cohen, Irvin	Sinai Hospital, Baltimore, Md.
Cooper, Harry Douglas	South Baltimore General Hospital, Baltimore, Md.
Cooper, William Benjamin, Jr.	South Baltimore General Hospital, Baltimore, Md.
Corzine, William Jenkins, Jr.	Roper Hospital, Charleston, S. C.
Coultas, Elizabeth Louise	Hospital of the Woman's Medical College, Philadelphia, Pa.
Cushner, Irvin Morris	Sinai Hospital, Baltimore, Md.
Dorman, Parker Sturgis	Ancker Hospital, St. Paul, Minn.
Duvall, Robert Caywood, Jr.	University Hospital, Baltimore, Md.
Eppes, Williford	University Hospitals, Iowa City, Iowa
[2]Evans, John Ebenezer, Jr.	University Hospital, Baltimore, Md.
Faraino, Frank Anthony	Mercy Hospital, Baltimore, Md.
Fink, Joel Charles	United States Army Internship
Fisher, Donald Ernst	Church Home & Hospital, Baltimore, Md.
Fisher, George William	Bon Secours Hospital, Baltimore, Md.
Fitzgerald, Robert Greeson, Jr.	Watts Hospital, Durham, N. C.
Forrer, Gordon Randolph	United States Marine Hospital, Baltimore, Md.
Friedman, Asher Arthur	Sinai Hospital, Baltimore, Md.
[2]Gakenheimer, William Alfred	University Hospital, Baltimore, Md.
Gardner, Robert Kline	Philadelphia General Hospital, Philadelphia, Pa.
Geddes, David Kofoid	Queen's Hospital, Honolulu, Hawaii
[1]Gerlach, Jane Knowles	Research Assistant, United States Public Health Service, Bethesda, Md.
Gillson, Edward Michael	St. Joseph Hospital, Paterson, N. J.
[2]Gold, Benjamin Miller	University Hospital, Baltimore, Md.
Grau, Edward Gordon	St. Joseph's Hospital, Baltimore, Md.
Grossman, Louis Ward, Jr.	Hospital of the Protestant Episcopal Church, Philadelphia, Pa.
Haase, Frederick Robert	Church Home and Hospital, Baltimore, Md.
[2]Hahn, Robert Roland	University Hospital, Baltimore, Md.

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[1] Research, not an internship.

[2] July 1, 1947 to June 30, 1949

Hall, Howard Essex	University Hospital, Baltimore, Md.
Hearne, Calvin Beck	University Hospital, Baltimore, Md.
Hightower, John Allen	University Hospital, Baltimore, Md.
Hogan, John Francis, Jr.	Union Memorial Hospital, Baltimore, Md.
Hooks, Richard Eugene	James Walker Memorial Hospital, Wilmington, N. C.
Houghton, James Francis	Jersey City Hospital, Jersey City, N. J.
[2]Hunter, Robert Carl	University Hospital, Baltimore, Md.
Imbrie, David Ellwood	Mercy Hospital, Pittsburgh, Pa.
Jernigan, Lane McDermott	Mercy Hospital, Baltimore, Md.
Judd, Allyn Francis	South Baltimore General Hospital, Baltimore, Md.
Leung, Bernard	Fordham Hospital, New York, N. Y.
Levin, Norman	West Baltimore General Hospital, Baltimore, Md.
LiPira, Joseph Francis	Mercy Hospital, Baltimore, Md.
Machata, Francis Karl	Charles S. Wilson Memorial Hospital, Johnson City, N. Y.
Mannino, Peter Paul	Bon Secours Hospital, Baltimore, Md.
[2]Mansberger, Arlie Roland, Jr.	University Hospital, Baltimore, Md.
Mattern, Carl Frederick Theodore	Garfield Memorial Hospital, Washington, D. C.
Mattern, Elizabeth Anne Dentry	Garfield Memorial Hospital, Washington, D. C.
Mitchell, Wallace Herttell	James M. Jackson Memorial Hospital, Miami, Fla.
Morrow, John Gordon, Jr.	Maryland General Hospital, Baltimore, Md.
Nachlas, Nathan Edward	Sinai Hospital, Baltimore, Md.
Pertz, Elden Hobart	Mercy Hospital, Baltimore, Md.
Phillips, David Lee	University Hospital, Baltimore, Md.
[2]Piggott, John Burr, Jr. B.S.	University Hospital, Baltimore, Md.
Post, William Reyman	Mercy Hospital, Baltimore, Md.
Powell, Jack Hanson, Jr.	University Hospital, Baltimore, Md.
Rasch, George Chesley, Jr.	Indiana University Medical Center, Indianapolis, Ind.
Ribet, James Ernest	Hospital of the Protestant Episcopal Church, Philadelphia, Pa.
Rogers, Joseph Megeath	Gallinger Municipal Hospital, Washington, D. C.
[2]Rosser, John Hays	University Hospital, Baltimore, Md.
Sachs, Sidney Herman	University Hospital, Baltimore, Md.
Salvati, Eugene Philip	Muhlenberg Hospital, Plainfield, N. J.
Schapiro, Joseph Solomon	Michael Reese Hospital, Chicago, Ill.
Schnitzker, William Frederic	Mercy Hospital, Baltimore, Md.
Scott, Harry Baty	Mercy Hospital, Baltimore, Md.
Shaw, John Hill	St. Agnes' Hospital, Baltimore, Md.
[2]Shear, Joseph	University Hospital, Baltimore, Md.
Shulman, Melvin	Queens General Hospital, Jamaica, N. Y.
Silber, Earle	Michael Reese Hospital, Chicago, Ill.
Smith, John Richard, Jr.	Union Memorial Hospital, Baltimore, Md.
Spino, Pascal Daniel	University Pittsburgh Medical Center, Pittsburgh, Pa.
Stenstrom, William Harold	Augustana Hospital, Chicago, Ill.
Sussman, Harold	Sinai Hospital, Baltimore, Md.
Swink, Robert Landrum	James M. Jackson Memorial Hospital, Miami, Florida
Trench, James McKechan	St. Francis Hospital, Pittsburgh, Pa.
Valderas, Jose Gervasio	St. Agnes' Hospital, Baltimore, Md.
Venable, Sidney Johnson	St. Agnes' Hospital, Baltimore, Md.
Waltz, Robert Claude	University Hospitals, Cleveland, Ohio
[2]White, John Phillips, III	University Hospital, Baltimore, Md.
Williams, James Delbert	Hospital of the Good Samaritan, Los Angeles, Calif.
Williams, John Elbert	Hospital of the Good Samaritan, Los Angeles, Calif.
Winokur, George	Church Home and Hospital, Baltimore, Md.
Zinberg, Norman Earl	Sinai Hospital, Baltimore, Md.

MATRICULATES

SENIOR CLASS, SEPTEMBER 5, 1946 TO JUNE 7, 1947

Abell, Harry Davis, *The Johns Hopkins University*........................Kentucky
Allen, Gladys Martha, *B.S., University of Maryland, 1943*................Maryland
Anthony, James Elmer, Jr., *B.S., Washington College, 1944*................Maryland
Beatty, John Orville, *B.S., University of Nevada, 1945*...................Nevada
Benson, John Fisher, *B.S., University of Maryland, 1945*..................Maryland
Berggreen, Raymond George, *A.B., Carleton College, 1943*................Minnesota
Bindeman, William Wylie, *B.S., Wheaton College, 1944*...............North Carolina
Blair, James Seaborn, Jr., *University of North Carolina; University of North Carolina School of Medicine, December 1943–June 1945*............North Carolina
Blevins, Joseph Wendell, *B.S., Geneva College, 1943*..................Pennsylvania
Brickner, John George, III, *B.S., University of Maryland, 1944*............Maryland
Bunch, Littleton Jay, *A.B., University of North Carolina, 1943; University of North Carolina School of Medicine, December 1943–June 1945*............North Carolina
Bunn, David Glenn, *University of North Carolina; University of North Carolina School of Medicine, December 1943–June 1945*....................North Carolina
Carouge, Gilbert Morris, *University of Maryland*........................Maryland
Chase, Henry Vincent, *Ph.B., Loyola College, 1943*........................Maryland
Coffey, Michael Joseph, *B.S., University of Notre Dame, 1945*.............New Jersey
Cohen, Bernard Stanley, *University of Maryland*..........................Maryland
Cohen, Irvin, *A.B., The Johns Hopkins University, 1944*....................Maryland
Cooper, Harry Douglas, *B.S., Washington College, 1941*....................Maryland
Cooper, William Benjamin, Jr., *B.S., University of Maryland, 1945*..........Maryland
Corzine, William Jenkins, Jr., *The Citadel*........................North Carolina
Coultas, Elizabeth Louise, *A.B., Goucher College, 1944*..................New Jersey
Cushner, Irvin Morris, *University of Maryland*...........................Maryland
Dorman, Parker Sturgis, *A.B., Amherst College, 1943*.............District of Columbia
Duvall, Robert Caywood, Jr., *University of Maryland*.....................Maryland
Eppes, Williford, *B.S., University of Maryland, 1945*.....................Maryland
Evans, John Ebenezer, Jr., *B.S., Davidson College, 1943*..............North Carolina
Faraino, Frank Anthony, *B.S., Loyola College, 1943*......................Maryland
Fink, Joel Charles, *B.S., University of Alabama, 1943; Medical College of Alabama, December 1943–May 1945*..Alabama
Fisher, Donald Ernst, *University of Maryland*............................Maryland
Fisher, George William, *B.S., Franklin and Marshall College, 1943*.........Maryland
Fitzgerald, Robert Greeson, Jr., *B.S. in Medicine, University of North Carolina, 1944, University of North Carolina School of Medicine, December 1943–June 1945* North Carolina
Forrer, Gordon Randolph, *B.S., University of Maryland, 1945*.............Maryland
Friedman, Asher Authur, *A.B., The Johns Hopkins University, 1943*........Virginia
Gakenheimer, William Alfred, *The Johns Hopkins University*.............Maryland
Gardner, Robert Kline, *University of Maryland*..........................Maryland
Geddes, David Kofoid, *A.B., University of California, 1946*..............California
Gerlach, Jane Knowles, *A.B., Colorado College, 1943*.....................Maryland
Gillson, Edward Michael, *A.B., St. Peter's College, 1943*................New Jersey
Gold, Benjamin Miller, *University of North Carolina; University of North Carolina School of Medicine, December 1943–June 1945*.................North Carolina
Grau, Edward Gordon, *B.S., Loyola College, 1943*.........................Maryland
Grossman, Louis Ward, Jr., *Westminster College*......................Pennsylvania
Haase, Frederick Robert, *University of Maryland*........................Maryland
Hahn, Robert Roland, *University of Maryland*............................Maryland
Hall, Howard Essex, *Western Maryland College*...........................Maryland
Hearne, Calvin Beck, *College of Wooster*....................................Ohio
Hightower, John Allen, *Emory University, University of North Carolina School of Medicine, December 1943–June 1945*..............................Florida
Hogan, John Francis, Jr., *Loyola College*................................Maryland
Hooks, Richard Eugene, *University of North Carolina; University of North Carolina School of Medicine, December 1943–June 1945*....................North Carolina
Houghton, James Francis, *Dartmouth College*...........................New Jersey
Hunter, Robert Carl, *B.S., University of Maryland, 1945*..................Maryland
Imbrie, David Ellwood, *A.B., Washington and Jefferson College*..........Pennsylvania

Jernigan, Lane McDermott, *University of Maryland*........Maryland
Judd, Allyn Francis, *A.B., Tufts College, 1944*........Massachusetts
Leung, Bernard, *B.S., University of Maryland, 1945*........New York
Levin, Norman, *University of Maryland*........Maryland
LiPira, Joseph Francis, *B.S., University of Maryland, 1945*........Maryland
Machata, Francis Karl, *Ph.B., Loyola College, 1943*........New York
Mannino, Peter Paul, *Western Maryland College*........Maryland
Mansberger, Arlie Roland, Jr., *Western Maryland College*........West Virginia
Mattern, Carl Frederick Theodore, *The Johns Hopkins University*........Maryland
Mattern, Elizabeth Anne Gordon Dentry, *A.B., College of Notre Dame of Maryland, 1943*........Maryland
Mitchell, Wallace Herttell, *B.S., University of Florida, 1943*........Florida
Morrow, John Gordon, Jr., *Emory University*........Georgia
Nachlas, Nathan Edward, *University of Maryland*........Maryland
Pertz, Elden Hobart, *B.S., West Virginia Wesleyan College, 1947*........West Virginia
Phillips, David Lee, *A.B., The Johns Hopkins University, 1943*........Maryland
Piggott, John Burr, Jr., *B.S., Virginia Military Institute, 1943*...District of Columbia
Post, William Reyman, *A.B., University of Mississippi, 1946*........Mississippi
Powell, Jack Hanson, Jr., *A.B., Emory University, 1943*........Georgia
Rasch, George Chesley, Jr., *B.S., University of Maryland, 1945*........Maryland
Ribet, James Ernest, *University of North Carolina; University of North Carolina School of Medicine, December 1943–June 1945*........North Carolina
Rogers, Joseph Megeath, *University of Maryland*........Virginia
Rosser, John Hays, *B.S., Wheaton College, 1943*........Maryland
Sachs, Sidney Herman, *University of Maryland*........Maryland
Salvati, Eugene Philip, *A.B., West Virginia University, 1944; West Virginia University School of Medicine, January 1944–June 1945*........West Virginia
Schapiro, Joseph Solomon, *The Johns Hopkins University*........Maryland
Schnitzker, William Frederic, *Transylvania College*........Maryland
Scott, Harry Baty, *University of Virginia*........Virginia
Senning, Frederick Stephen, *University of Maryland*........Maryland
Shaw, John Hill, *B.S., Hampden-Sydney College, 1943*........Virginia
Shear, Joseph, *University of Maryland*........Maryland
Shulman, Melvin, *The Johns Hopkins University*........Maryland
Silber, Earle, *A.B., The Johns Hopkins University, 1944*........Maryland
Smith, John Richard, Jr., *B.S., Washington College, 1943*........Maryland
Spino, Pascal Daniel, *Bethany College*........Pennsylvania
Stenstrom, William Harold, *B.S., College of Puget Sound, 1943*........Washington
Sussman, Harold, *A.B., The Johns Hopkins University, 1943*........Maryland
Swink, Robert Landrum, *Emory University*........Florida
Trench, James McKechan, *B.S., Bethany College, 1945*........Pennsylvania
Valderas, Jose Gervasio, *University of Puerto Rico*........Puerto Rico
Venable, Sidney Johnson, Jr., *B.S., Hampden-Sydney College, 1943*........Maryland
Waltz, Robert Claude, *B.S., Washington & Jefferson College, 1943*........Ohio
White, John Phillips, III, *The Johns Hopkins University*........Maryland
Williams, James Delbert, *Texas Technological College*........California
Williams, John Elbert, *Texas Technological College*........California
Winokur, George, *A.B., The Johns Hopkins University, 1944*........Maryland
Zinberg, Norman Earl, *B.S., University of Maryland, 1942*........Maryland

JUNIOR CLASS, SEPTEMBER 5, 1946 TO JUNE 7, 1947

Alecce, Angelo Andrew, *B.S., Loyola College, 1945*........Maryland
Allison, George Malcolm, *Marquette University*........Illinois
Aponte, Joseph Louis, *University of Puerto Rico*........Puerto Rico
Auld, David, *Swarthmore College*........Maryland
Baker, Frank William, Jr., *University of Chicago*........Maryland
Beyer, David Henry, *Cornell University*........Minnesota
Bisanar, James Milton, *Davidson College*........North Carolina
Bond, Donald Buckey, *A.B., Western Maryland College, 1938*........Maryland
Bronstein, Eugene Leonard, *Emory University*........New York
Buchness, John Michael, *A.B., Loyola College, 1944*........Maryland
Bullock, John Boyd, *University of Richmond*........Virginia
Bulluck, Matt Heyer, *Guilford College*........North Carolina

Chaimovitz, Robert, *Bethany College*	Pennsylvania
Chelton, Alice Katherine Graybill, *University of Chicago*	West Virginia
Condron, James Frank, *Muhlenberg College*	Pennsylvania
Crecraft, Harold James, *A.B., Taylor University, 1944*	Pennsylvania
Dalton, James Breckenridge, Jr., *University of Richmond*	Virginia
Dempsey, Raymond Joseph, *B.S., University of Notre Dame, 1944*	Illinois
Dwyer, Frank Philip, Jr., *Mt. St. Mary's College*	Pennsylvania
Ensor, Robert Ellwood, *Western Maryland College*	Maryland
[4]Fravel, Charles Richard, *Cornell University*	Virginia
Golombek, Leonard Harry, *University of Maryland*	Maryland
Grant, Bowie Linn, *Potomac State School of West Virginia*	Maryland
Green, James William, *Western Maryland College*	Maryland
Hamrick, George Vincent, *Washington University*	Maryland
Hankins, John Roland, *University of Virginia*	Virginia
Heldrich, Frederick Joseph, Jr., *A.B., Gettysburg College, 1945*	Maryland
Hoback, Florence Kunst, *A.B., West Virginia University, 1944*	West Virginia
Hobart, Richard Loren, Jr., *Washington and Lee University*	District of Columbia
Holloway, William Jackson, *Western Maryland College*	Maryland
Insley, Marion Clarence, Jr., *University of Maryland*	Maryland
Kastner, Lee Norman, *University of Pennsylvania*	Maryland
Kaufman, Raymond Henry, *University of North Carolina*	New York
Kemp, Katherine Virdin, *A.B., College of Notre Dame of Maryland, 1943*	Maryland
Kennedy, Carl Hubert, Jr., *University of Virginia*	Pennsylvania
Koch, Lillian Dorothy, *B.S., University of Maryland, 1944*	Maryland
Leograndis, Stephen C., *Duke University*	Massachusetts
Lithgow, Charles Henry, *Loyola College*	Maryland
Mack, Harry Patterson, *Washington University*	New York
Mallis, Nicholas, *Cornell University*	Maryland
Matthews, Burton Vincent, *A.B., The Johns Hopkins University, 1943; Cornell University*	Maryland
Matthews, Roland Dellwood, *University of North Carolina; University of North Carolina School of Medicine, September 1944–March 1946*	North Carolina
McCauley, Betty Jane, *B.S., University of Maryland, 1943*	Maryland
McCrumb, Fred Rodgers, Jr., *Loyola College*	Maryland
Mohler, Donald Ignatius, Jr., *Loyola College*	Maryland
Newell, Edward Alphonso, *Western Maryland College*	Maryland
Niswander, Guy Donald, *University of Michigan*	West Virginias
Nolan, Paul Vernon, *B.S. in Medicine, University of North Carolina, 1945; University of North Carolina School of Medicine, September 1944–March 1946*	North Carolina
Padussis, Stephen Konstant, *University of Maryland*	Maryland
Petersen, Phyllis Ann, *A.B., University of Michigan, 1943*	Maryland
Platt, Julian Jay, *Loyola College*	Maryland
Powell, Albert Milton, Jr., *University of North Carolina*	Maryland
Redding, Joseph Stafford, *A.B., University of North Carolina, 1943; University of North Carolina School of Medicine, September 1944–March 1946*	North Carolina
Rhyne, Jimmie Lee, *University of North Carolina*	North Carolina
Rodriguez, Edsel Antonio, *College of the Holy Cross*	Puerto Rico
Rudolph, Robert Lee, *Ohio University*	Ohio
Sanford, William Gordon, *University of North Carolina, University of North Carolina School of Medicine, September 1944–March 1946*	North Carolina
Scherr, Merle Sundrell, *West Virginia University*	West Virginia
Schwartz, Benson Charles, *University of Maryland*	Maryland
Shell, John Robert, *University of Richmond*	Virginia
Silverman, Benjamin K., *A.B., The Johns Hopkins University, 1947*	Maryland
Silverstein, Daniel Lewis, *Loyola College*	Maryland
Siwinski, Thaddeus Charles, *A.B., Loyola College, 1944*	Maryland
Smelser, Rennert Marquette, *University of Maryland*	Maryland
Snyder, William Allen, *The Johns Hopkins University*	Maryland
Stahl, Robert Ray, *University of New Hampshire*	Pennsylvania
Swisher, Kyle Young, Jr., *Bethany College*	West Virginia

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[4] Did not complete the year—illness.

Tarr, Norman, *Washington College*	Maryland
Tate, Allen Denny, Jr., *B.S. in Medicine, University of North Carolina, 1945; University of North Carolina School of Medicine, September 1944–March 1946*	North Carolina
Theuerkauf, Frank Joseph, Jr., *Villa Maria College*	Pennsylvania
Thomas, Clyde Dana, Jr., *Bethany College*	West Virginia
Thuss, William Getz, Jr., *Emory University*	Alabama
Walters, Hezekiah G., Jr., *A.B., University of North Carolina, 1944*	North Carolina
Ware, Charles Ingersoll, *Wake Forest College*	New Jersey
Waterman, Roger Sherman, *Bethany College*	Pennsylvania
Welborn, James Todd, *B.S., Davidson College, 1944; University of North Carolina School of Medicine, September 1944–March 1946*	North Carolina
Whitehorn, Clark Allan, *The Johns Hopkins University*	Michigan
Wilson, John Dean, *Bethany College*	Pennsylvania
Womack, William Spengler, *Emory and Henry College*	Virginia
Young, John Paul, *The Johns Hopkins University*	Maryland

SOPHOMORE CLASS, SEPTEMBER 5, 1946 TO JUNE 7, 1947

Abraham, Robert Auman, *B.S., Franklin and Marshall College, 1945*	Pennsylvania
Bachman, Leonard, *B.S., Franklin and Marshall College, 1946*	Maryland
[2]Barlow, Lundie Weathers, Jr., *University of Minnesota*	Virginia
Barnard, John William, *B.S., Milligan College, 1937*	Maryland
Belkin, Joseph William, *College of The Holy Cross*	Connecticut
Benjamin, William Philip, *A.B., Brooklyn College, 1943*	New York
Blundell, Albert Edward, *Temple University*	Utah
Cline, James Alexander, III, *University of Pennsylvania*	Nebraska
[2]Collings, Harold, Jr., *The Johns Hopkins University*	Indiana
Deringer, Florence Hurtt, *B.S., Washington College, 1945*	Maryland
Ensor, Wilmer Clifton, Jr., *B.S., Loyola College, 1945*	Maryland
Eubanks, Otha Albert, Jr., *Duke University*	North Carolina
Forman, Robert Blaine, *University of Oregon*	Oregon
Gill, Joseph Edward, *Union College*	Pennsylvania
[4]Goldstein, Arthur Jay, *Bethany College*	New York
Gorten, Martin Klaus, *A.B., Western Maryland College, 1943*	New Jersey
Gray, Harry Williams, *Duke University*	Maryland
Harris, James Radcliffe, *Temple University*	Pennsylvania
Henderson, Charles Thomas, *The Johns Hopkins University*	Illinois
Humphreys, Charles Wesley, Jr., *Duke University*	District of Columbia
Knabe, George William, Jr., *The Johns Hopkins University*	Michigan
[3]Krieger, John Louis, *A.B., St. Louis University, 1943*	Missouri
Lewis, Thomas Earl, *Pennsylvania State College*	Pennsylvania
Lock, Burton Vernon, *The Johns Hopkins University*	Maryland
London, Nathaniel Jacob, *The Johns Hopkins University*	Maryland
Longley, George Henry, *University of Minnesota*	Maryland
Lukens, James Thomas, *Vanderbilt University*	Pennsylvania
Malcarney, Alberta Rose, *A.B., University of Southern California, 1944*	California
Mattax, Harry McCoy, *Western Maryland College*	Maryland
May, Homer Woodrow, *B.S., University of Pittsburgh, 1944*	Pennsylvania
McCord, Charles Frederic, *Pennsylvania State College*	Indiana
Medairy, Mary Veronica, *A.B., College of Notre Dame of Maryland, 1943*	Maryland
[3]Metcalf, John Shelby, Jr., *University of California*	California
Middleton, Edmund Bishop, *The Johns Hopkins University*	Rhode Island
Miller, Max Jay, *University of Richmond*	Maryland
Moyer, John Lewis, III, *University of Minnesota*	Pennsylvania
Neumayer, Francis, *University of Maryland*	Pennsylvania
Newell, Josephine Evelyn, *University of the State of South Carolina*	North Carolina
Nicklas, Gilbert Lee, *B.S., Franklin and Marshall College, 1946*	Maryland
Panzarella, John Henry, *Fordham University*	New York

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[2] Withdrew after satisfactory completion of year.
[3] Withdrew August 30, 1946 in good standing.
[4] Did not complete the year—illness.

Parelhoff, Merrill Elliott, *University of Maryland*....................Maryland
Pleet, Jerome, *Stanford University, University of Texas School of Medicine, September 1945–May 1946*....................Maryland
Raskin, Howard Frank, *The Johns Hopkins University*....................Maryland
Reisch, Rashi Schorr, *B.S., University of Miami, Florida, 1944*..............Florida
Riddell, Clifford Thurston, Jr., *A.B., Bridgewater College, 1933*............Virginia
Roehrig, Charles Burns, *Vanderbilt University*....................Massachusetts
Sarewitz, Albert Bernard, *University of Pennsylvania*..................New Jersey
Scher, Jordan Mayer, *A.B., Wesleyan University, 1946*...................Maryland
Schmale, Arthur Henry, Jr., *Pennsylvania State College*...................Nebraska
Schnaper, Nathan, *B.S., Washington College, 1940*.....................Maryland
Schreiber, Richard David, *Bethany College*.............................Illinois
Sherrard, Margaret Lee, *A.B., Seton Hill College, 1945*..............West Virginia
Smith, Meredith Parks, *St. Lawrence University*......................Minnesota
Snider, Kenneth Bruce, *University of Maryland*..........................Texas
Spittell, John Allen, Jr., *B.S., Franklin and Marshall College, 1944*........Maryland
Stahler, Earlin John, *University of Maryland*.......................Pennsylvania
Stanfield, Elwin Eugene, *University of Pennsylvania*...................California
Steckler, Robert Joseph, *A.B., Evansville College, 1943*...................Indiana
Stevenson, Edward Ward, *Duke University*............................Virginia
Strahan, John Franklin, *Duke University*............................Maryland
Tilley, Russell McFarlane, Jr., *University of Rochester*..................New York
Timanus, James Kyner, *Oberlin College*................................Ohio
Tramer, Arnold, *University of Maryland*.............................Maryland
Trettin, Gene Douglas, *Ursinus College*.............................Maryland
Warnock, Robert George, *Bates College*...............................Ohio
Watson, Frank Yandle, *Duke University*....................... North Carolina
Welch, Elbert Sylvester, *Duke University*............................Alabama
Wolfe, Carolyn DeWitt, *A.B., Hollins College, 1945*...................New Jersey
Ziegler, John Bosley, *A.B., Gettysburg College, 1942*....................Maryland

FRESHMAN CLASS, SEPTEMBER 5, 1946 TO JUNE 7, 1947

Andersen, William Andrew, *The Johns Hopkins University*................Maryland
Bacon, John Louis, *Wagner College*.................................New York
Bagley, Charles, *B.S., Loyola College, 1945*...........................Maryland
Baumann, Wilbur Nelson, *University of Maryland*.......................Maryland
Bisgyer, Jay Lewis, *University of Pennsylvania*..................District of Columbia
Bleecker, Harry Harlan, Jr., *University of California*..................California
Borges, Francis Joseph, *University of Maryland*.........................Maryland
Bradshaw, Raymond, Jr., *B.S., University of Maryland, 1943*...............Maryland
Bronushas, Joseph Benedict B., *B.S., Loyola College, 1946*................Maryland
[1]Brown, Lorence Francis, *B.S., West Virginia Wesleyan College, 1943*...West Virginia
Burkey, Fred Jamison, *St. Francis College*............................Maryland
Chelton, Louis Guy, *Gettysburg College*..............................Maryland
Coller, Jerome Joel, *A.B., The Johns Hopkins University, 1946*............Maryland
Corpenning, Tom Nye, *University of North Carolina*................North Carolina
Cowen, Joseph Robert, *The Johns Hopkins University*....................Maryland
Cracraft, William Atkinson, *B.S., Virginia Military Institute, 1939*....West Virginia
[1]Cullison, Robert Morton, *A.B., University of Iowa, 1942*................Maryland
Daly, Harold Lawrence, Jr., *University of Maryland*.....................Maryland
Deitz, Leonard Loeb, *A.B., University of North Carolina, 1946*.............Maryland
Demarest, Lawrence Mann, *B.S., Bowdoin College, 1945*.................New York
Demmy, Nicholas, *B.S., University of Pittsburgh, 1942*.................Pennsylvania
Edmunds, Frederick Thomas, *B.S., Hampden-Sydney College, 1941*......West Virginia
Farson, deCamp Butler, *A.B., Goucher College, 1942*......................Maryland
[5]Golob, Charles George, *B.S., Pennsylvania State College, 1940*............Maryland
Googins, Charles George, *B.S., University of Maryland*...................Kansas
Gould, Virginia Marie, *B.S., Nazareth College, 1941*....................New York
Greenstein, George Herbert, *A.B., The Johns Hopkins University, 1941*...Maryland

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[1] Did not complete the year.
[5] Withdrew November 11, 1946, in good standing.

Hamberry, Leonard Gerard, *A.B., Loyola College, 1940*	Maryland
Hawkins, George Kenneth, *B.S., Upsala College, 1943*	New Jersey
Healy, John Clauson, *University of Connecticut*	Connecticut
Henson, Stanley Willard, Jr., *B.S., Oklahoma A. & M. College, 1945*	Maryland
Heuman, Philip Walter, *B.S., University of Michigan, 1941*	Maryland
Hofsteter, Grace, *College of Wooster*	Ohio
[6]Horowitz, Leon David, *B.S., Washington College*	Maryland
Hoyt, Irvin Gorman, *A.B., St. Johns College, Annapolis, 1939*	Maryland
Huffer, Sarah Virginia, *B.S., University of Maryland, 1940*	Maryland
Husted, Harriet Lamont, *A.B., Wells College, 1944*	New Jersey
Hyle, John Charles, *B.S., Loyola College, 1942*	Maryland
Ibsen, Maxwell, *A.B., The Johns Hopkins University, 1939; A.M., University of Pennsylvania, 1940*	Pennsylvania
Iten, George Joseph, *A.B., Goshen College, 1946*	California
Jenkins, Sarah Anetta, *B.S., George Washington University, 1946*	Maryland
Jensen, Roy David, *B.S., University of Nevada, 1941*	Nevada
[1]Jones, William Irving, Jr., *B.S., Washington College, 1943*	Maryland
Kasik, Frank Thomas, *University of Maryland*	Maryland
Kelley, Gordon William, *University of Maryland*	Maryland
[1]Klitenic, Simon, *University of Maryland*	Maryland
[1]Knight, William Ira Charles, *B.S., Elizabethtown College, 1946*	Pennsylvania
Lewis, Thomas Franklin, Jr., *A.B., Western Maryland College, 1941*	Maryland
Lichtenberg, Joseph David, *A.B., The Johns Hopkins University, 1943*	Maryland
Martin, George William, Jr., *University of Delaware*	Delaware
McElvain, Wilbert Harding, *B.S., Grove City College, 1943*	Pennsylvania
[1]Meese, Billie Getson, *University of Akron*	Maryland
Miller, Dorothea Anna, *B.S., Washington College, 1946*	Maryland
Miller, Robert Eugene, *A.B., Bridgewater College, 1943*	Maryland
Morenz, Gloria Elizabeth, *A.B., The College of Notre Dame, 1946*	Maryland
Myers, Evangeline, *A.B., West Virginia University, 1945; A.M., Columbia University, 1946*	West Virginia
Noguera, Julio Tomas, *University of Puerto Rico*	Puerto Rico
O'Malley, Joseph Edward, *Colby College*	Maryland
Rver, William Benjamin, Jr., *Cornell University*	Maryland
Reynaud, Louis Favrot, Jr., *Emory University*	Illinois
Richardson, Paul Frederick, *Baylor University*	Maryland
Righetti, Milton Raymond, *University of California*	California
Roth, Oliver Ralph, *University of Maryland*	Maryland
Rubin, Seymour Howard, *A.B., The Johns Hopkins University, 1946*	Maryland
Rudy, Norman Edward, *Fresno State College*	California
Sandler, Robert, *B.S., University of Maryland, 1943*	Maryland
Shamer, Miriam Abbott, *A.B., Goucher College, 1946*	Maryland
Shepherd, Frederick Parker, *A.B., Syracuse University, 1935*	Maryland
Simmons, Frederic Rudolph, Jr., *B.S., Loyola College, 1946*	Maryland
Sires, William Oscar, *Western Maryland College*	Maryland
Sklar, Allen Leon, *Western Maryland College*	Maryland
Smith, Boylston Dandridge, Jr., *West Virginia University*	West Virginia
Smith, Morton, *University of Maryland*	Maryland
Sosnowski, Andrew Raymond, *B.S., Loyola College, 1945*	Maryland
[1]Spanier, Allen Benson, *B.S., John B. Stetson University, 1946*	Florida
Spaulding, Raymond Charles, Jr., *B.S., John B. Stetson University, 1943*	Florida
Storm, Mary Elizabeth, *A.B., Swarthmore College, 1946*	Maryland
Sulka, Casimir Michael, *Loyola College*	Illinois
Thibadeau, Robert Torndorff, *B.S., University of Maryland, 1945*	Maryland
Thompson, William Wesley, *B.S., Washington College, 1938*	Maryland
Upton, Albert Louis, *A.B., University of California, 1942*	California
Van Goor, Kornelius, *Calvin College*	New Jersey
Vicens, Enrique A., *University of Puerto Rico*	Puerto Rico
[1]Vincent, William Gerald, *A.B., Western Maryland College, 1942*	Maryland
Weed, Elinor Coolidge, *A.B., Randolph-Macon Woman's College, 1946*	Maryland

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[1] Did not complete the year.

[6] Withdrew November 11, 1946 in good standing.

White, Fowler Felix, *B.S., Trinity College, 1942*	Connecticut
[4]Williams, Martin Shelton, *University of Virginia*	Virginia
Wilson, Clifford Edward, Jr., *Bowdoin College*	Connecticut
Wolf, Ernest Simon, *University of Maryland*	Maryland
Yeager, William Howard, Jr., *B.S., University of Maryland, 1944*	Maryland

SPECIAL STUDENTS

Alice Ruth Bentz, *B.S., University of Maryland, 1943*	Maryland

STUDENTS IN ART AS APPLIED TO MEDICINE

Margaret Wetherill Wood, *R.N.*	Maryland

POSTGRADUATE STUDENTS

July 1, 1946 to June 30, 1947

Surgical and Systemic Anatomy of Ear, Nose and Throat

	Medical School
Morris Edward Krucoff, M.D.	*George Washington University*
Clyde G. Sussman, M.D.	*University of Indiana*

Surgical Anatomy "B"

Edgar Berman, M.D.	*University of Maryland*
Charles Gallion, M.D.	*College of Medical Evangelists*
Milton Ginsberg, M.D.	*University of Maryland*
Jacob Katz, M.D.	*New York University*
Arthur W. Lyons, M.D.	*Tufts College Medical School*
Alfred F. Schmidt, M.D.	*Kansas University*
Paul E. Walker, M.D.	*University of Tennessee*

Refresher Course in General Medicine and Surgery

Richard L. Fowler, M.D.	*University of Maryland*
Georgia Funtanellas, M.D.	*University of Havana*
Helene D. Michel, M.D.	*Johns Hopkins University*
John C. Osborne, M.D.	*University of Maryland*
Maurice I. Shub, M.D.	*University of Maryland*
Gertrude Sonnenfeldt, M.D.	*Friedrich Wilhelm University, Berlin, Germany*
Alfred W. Yocum, M.D.	*Baltimore Medical College*

Gynecology and Obstetrics "A"

Fay M. Nace, M.D.	*University of Oregon*
Keaciel Krulevitz, M.D.	*University of Maryland*

Gynecology, Oncology and Female Urology "B"

Fay M. Nace, M.D.	*University of Oregon*

Neuropathology

Sidney R. Govans, M.D.	*University of Maryland*
Jack R. Jarvie, M.D.	*Vanderbilt University Medical School*

Pathology "B" (Surgical)

Theodore Kardash, M.D.	*University of Maryland*
Robert Hilton Williams, M.D.	*University of Indiana*

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[4] Did not complete the year—illness.

PATHOLOGY (SPECIAL)

SAMUEL J. ARNOLD, M.D.	
MORRIS BURLESON, M.D.	*Duke University*
GABRIEL F. CUCOLO, M.D.	
CHARLES S. GALLION, M.D.	*College of Medical Evangelists*
MILTON GINSBERG, M.D.	*University of Maryland*
WILLIAM H. HEATH, M.D.	*University of Tennessee*
JACOB F. KATZ, M.D.	*New York University*
GEORGE W. SMITH, M.D.	*University of Indiana*
S. JAMES THOMISON, JR., M.D.	*New York University*

SUMMARY OF STUDENTS

September 5, 1946 to June 7, 1947

Medical Students	*Male*	*Female*	*Total*
Senior Class	93	4	97
Junior Class	74	6	80
Sophomore Class	62	7	69
Freshman Class	78	12	90
	307	29	336
Withdrawals	13		13
	294	29	323
Special students	0	1	1
Students in art as applied to medicine	0	1	1
	294	31	325

GEOGRAPHICAL DISTRIBUTION OF STUDENTS

September 5, 1946 to June 7, 1947

Alabama	3
California	11
Connecticut	4
Delaware	1
District of Columbia	5
Florida	6
Georgia	2
Illinois	6
Indiana	3
Kansas	1
Kentucky	1
Maryland	160
Massachusetts	3
Michigan	2
Minnesota	3
Mississippi	1
Missouri	1
Nebraska	2
Nevada	2
New Jersey	11
New York	12
North Carolina	24
Ohio	6
Oregon	1
Pennsylvania	27
Rhode Island	1
Texas	1
Utah	1
Virginia	14
Washington	1
West Virginia	15
UNITED STATES POSSESSIONS	
Puerto Rico	5

LIST OF COLLEGE SOURCES OF STUDENTS ENROLLED

September 5, 1946 to June 7, 1947

Akron, University of	1
Alabama, University of	1
Amherst College	1
Bates College	1
Baylor University	1
Bethany College	9
Bowdoin College	2
Bridgewater College	2
Brooklyn College	1
California, University of	5
Calvin College	1
Carleton College	1
Chicago, University of	2
Citadel, The	1
Colby College	1
Colorado College	1
Columbia University	1
Connecticut, University of	1
Cornell University	5
Dartmouth College	1
Davidson College	3
Delaware, University of	1
Duke University	8
Elizabethtown College	1
Emory University	7
Emory and Henry College	1
Evansville College	1
Florida, University of	1
Fordham University	1
Franklin & Marshall College	5
Fresno State College	1
Geneva College	1
George Washington University	1
Gettysburg College	3
Goshen College	1
Goucher College	3
Grove City College	1
Guilford College	1
Hampden-Sydney College	3
Hollins College	1
Holy Cross, College of The	2
Iowa, University of	1
Johns Hopkins University, The	31
Loras College	1
Loyola College	20
Marquette University	1
Maryland, University of	54
Miami, University of (Florida)	1
Michigan, University of	3
Milligan College	1
Minnesota, University of	3
Mississippi, University of	1
Mt. St. Mary's College	1
Muhlenberg College	1
Nazareth College	1
Nevada, University of	2
New Hampshire, University of	1
North Carolina, University of	18
Notre Dame of Maryland, College of	4
Notre Dame, University of	2
Oberlin College	1
Ohio University	1
Oklahoma A. & M. College	1
Oregon, University of	1
Pennsylvania State College	4
Pennsylvania, University of	6
Pittsburgh, University of	2
Potomac State School, W. Va	1
Puerto Rico, University of	4
Puget Sound, College of	1
Randolph-Macon Woman's College	1
Richmond, University of	4
Rochester, University of	1
St. Francis College	1
St. John's College, Annapolis	1
St. Lawrence University	1
St. Louis University	1
St. Peter's College	1
Seton Hill College	1
Southern California, University of	1
South Carolina, University of State of	1
Stanford University	1
Stetson University, John B.	2
Swarthmore College	2
Syracuse University	1
Taylor University	1
Temple University	2
Texas Technological College	2
Transylvania College	1
Trinity College	1
Tufts College	1
Union College	1
Upsala College	1
Ursinus College	1
Vanderbilt University	2
Villa Maria College	1
Virginia Military Institute	2
Virginia, University of	4
Wagner College	1
Wake Forest College	1
Washington College	10
Washington University	2
Washington & Jefferson College	2
Washington and Lee University	1
Wells College	1
Wesleyan University	1
Western Maryland College	14
Westminster College	1
West Virginia University	5
West Virginia Wesleyan College	2
Wheaton College	2
Wooster, College of	2

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(Term beginning July 1, 1947 and ending June 30, 1948)

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(Here state amount or describe property)

To School of Medicine

I give, devise and bequeath to the Regents of the University of Maryland, a corporation incorporated under the laws of the State of Maryland, for the benefit of the Faculty of Medicine...

(Here state amount or describe property)

THE UNIVERSITY OF MARYLAND SCHOOL OF NURSING

The University of Maryland School for Nurses was established in the year 1889. Since that time it has been an integral part of the University of Maryland, coming under the same government. It is a non-sectarian school, the only religious services being morning prayers.

The University Hospital is a general hospital containing about 435 beds. It is equipped to give young women a thorough course of instruction and practice in all phases of nursing.

Programs offered: The programs of study of the school are planned for two groups of students: (a) the five-year group, (b) the 36-month group.

The requirements for admission to the five-year program of the School of Nursing are the same as for other colleges of the University. The completion of this course entitles the student to the degree of Bachelor of Science from the University of Maryland and to the diploma of the University Hospital School of Nursing.

The requirements for admission to the 36-month program are the satisfactory completion of an academic course in an approved high school. The completion of this course entitles the student to the diploma of the University of Maryland School of Nursing.

Applications and catalogues may be obtained from the Director of the School of Nursing, University Hospital, Baltimore 1, Md.

MERCY HOSPITAL SCHOOL OF NURSING

The Mercy Hospital School of Nursing was established in 1899 and incorporated on December 23, 1901. It is an integral part of the Mercy Hospital and is under the same government. Mercy Hospital affords exceptional advantages for the education of nurses. It is a general hospital containing 348 beds, and offers opportunities for a thorough and varied experience. By its institutional affiliation with the University of Maryland it secures professors who give to the student the results of their training and experience in the fields of the medical and affiliated sciences.

The University of Maryland, in affiliation with the Mercy Hospital School of Nursing, offers a five year combined Academic and Nursing program. The completion of this course entitles the student to the degree of Bachelor of Science from the University of Maryland, and to the diploma of the Mercy Hospital School of Nursing. Graduate nurses who hold college degrees are greatly in demand, especially for positions in administration and teaching institutions. This program consequently offers a distinct advantage.

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[1] *Erratum*—Should read Charles Bagley, III on page 97.

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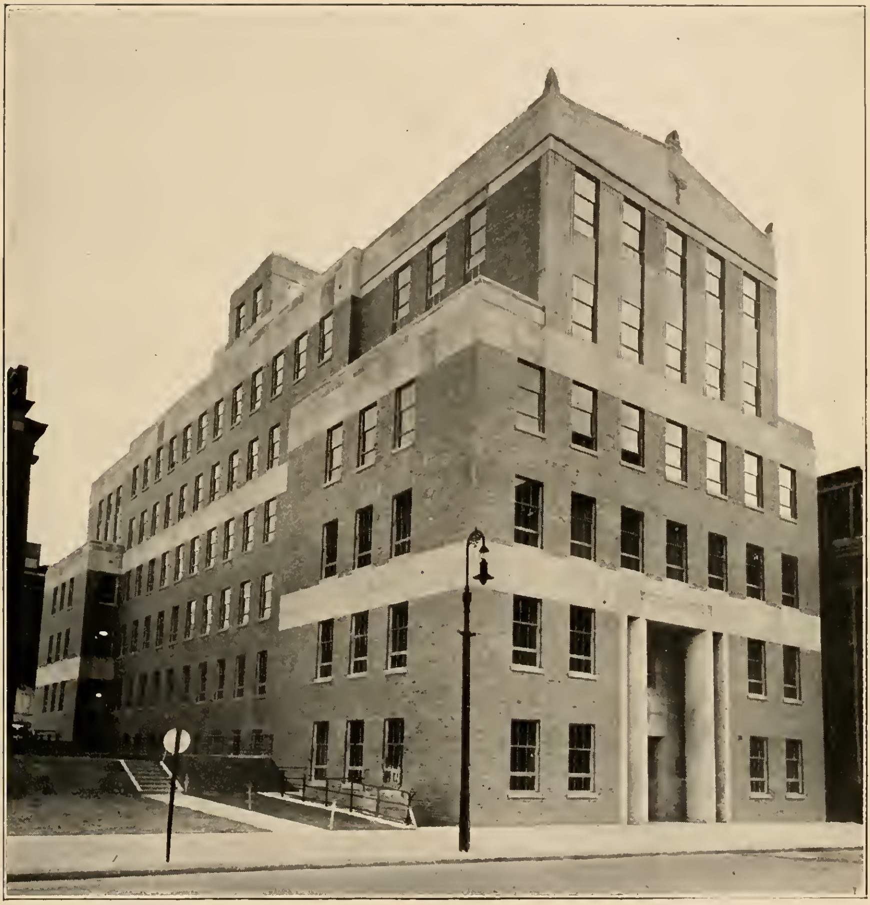

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MODERN CONCEPTS OF THE RECOGNITION AND TREATMENT OF CERTAIN ENDOCRINE DISORDERS*†

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Endocrinology embraces a wide field of disorders, attributable to aberrations in function of the various endocrine glands. In recent years, many of these disorders have been more clearly delineated as to pathogenesis, diagnosis and treatment. This article attempts to summarize present-day concepts of a number of these conditions, but it is not intended as a complete review of endocrinology. Endocrine disorders of childhood and gynecologic endocrinology have been omitted except where their inclusion was necessary for a complete description of a disease. In addition, hyperthyroidism is not discussed, as a report is forthcoming from this clinic on the use of thiourea derivatives in the treatment of this disorder.

ENDOCRINE DISEASES OF ADULTS

Thyroid. The thyroid gland is primarily concerned with the production of a hormone, the essential function of which is calorigenic, or the regulation of the rate of metabolism of every cell in the human body. The dwarfed congenitally hypothyroid child and the taller-than-average hyperthyroid child demonstrate the growth-promoting action of the hormone. Similarly, the non-pitting edema of hypothyroidism represents the retention of water, salts and protein which occurs with decreased amounts of circulating thyroid hormone (1), while many hyperthyroid patients show unusually prompt diuresis when given excess fluid by mouth (2). The effects of increased thyroid hormone on the nervous system are revealed in the emotional insta-

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bility, irritability, and occasional marked changes in cerebration which characterize hyperthyroidism. The same disease is frequently associated with varying degrees of muscular weakness as a manifestation of the increased mobilization and excretion of creatine stores (3). Aberrations in carbohydrate metabolism often accompany thyroid disease and are probably caused by a depletion of liver glycogen plus excessive glucose absorption from the gastrointestinal tract. A diabetic type of glucose tolerance curve is frequently seen in hyperthyroidism.

Hypothyroidism. The full blown clinical syndrome of thyroid deficiency is known as myxoedema or Gull's Disease. Myxoedema may occur as a spontaneous disease, or it may follow roentgen-ray therapy to the thyroid gland, iodine therapy, or surgery. At times it is associated with or follows thyroiditis; it may occur as a sequel to untreated hyperthyroidism, and is occasionally seen in patients whose thyroid glands show a nodular enlargement (4). It is more common in women than in men (4:1), and occurs most frequently during the fourth, fifth and sixth decades of life.

The outstanding manifestations of myxoedema are: (1) puffiness of the face and eye-lids, (2) swelling of the tongue and larynx producing a hoarse, slow, slurred speech, (3) dryness and roughness of the skin, (4) loss of hair, (5) mental lethargy, and (6) sensitivity to cold. Characteristically menstruation is excessive, but the patients are usually sterile.

Clinical laboratory data of aid in the diagnosis are: (1) a low basal metabolic rate (−20 per cent to −40 per cent), (2) a high blood cholesterol, (3) a secondary anemia, and (4) decreased excretion of the 17-keto-steroids. The frequency of an elevated blood cholesterol has been emphasized by many writers, and this determination should be obtained on any patients suspected of the disease. Other disorders characterized by an elevated blood cholesterol are: nephrosis, diabetes, pregnancy and chole-lithiasis (4). In cases of juvenile hypothyroidism, Wilkins and Fleischmann (5) have demonstrated that patients showing a borderline elevation of cholesterol treated with thyroid for three to six weeks will show a marked and diagnostic elevation of the level on withdrawal of the drug. This valuable test has not been investigated in adult hypothyroids but should produce a similar response.

A slow pulse, electrocardiographic changes and cardiac enlargement with failure may reflect the cardiovascular changes.

Myxoedema may be confused with chronic nephritis, pernicious anemia and panhypopituitarism. The appearance of the patient is especially suggestive of chronic nephritis.

The response of these patients to treatment with dessicated thyroid is usually so dramatic that in questionable cases a therapeutic test is indicated. Certain pertinent facts must be borne in mind: (1) Start with a small dose, ½ grain to 1 grain daily, and do not increase the dosage more often than every three weeks; (2) Always stipulate U.S.P. thyroid, as there are other preparations on the market which are of greater or less potency; (3) In treating patients with cardiovascular enlargement, electrocardiographic changes or any signs of coronary artery disease, start with $\frac{1}{4}$ grain or less daily and proceed cautiously. Large doses of dessicated thyroid may precipitate angina pectoris or coronary thrombosis; (4) Symptoms of thyroid overdosage include aching, muscle tenderness, fever, nausea and diarrhea, (5) Thyroxin possesses no advantages over dessicated thyroid and is more expensive.

The physician is constantly faced with the problem of the overweight patient who complains of fatiguability and sensitivity to cold. Basal metabolic rates in this group are frequently low, and the patient is led to believe from a survey of the more popular news and literary magazines that dessicated thyroid represents an easy cure for his or her problem. In these patients a careful search for stigmata of hypothyroidism is indicated, and several determinations of the blood cholesterol should be obtained. There is general agreement among those who have studied thyroid function that hypothyroidism is rarely a cause of obesity (4, 6). A family history of hyperthyroidism or the presence of goitre in these patients indicate caution in the use of thyroid, as Brunn (7) has recently reported a series of patients in whom hyperthyroidism developed after the treatment with thyroid preparation, and the patient should be advised that the use of these compounds is not without danger. If reasonable doubt as to the diagnosis exists, a therapeutic trial for a limited period is indicated with cessation of therapy if prompt alleviation of symptoms does not occur. When 3 grains of thyroid daily does not produce improvement, the patient probably does not have hypothyroidism.

Pituitary. A recapitulation of known pituitary functions should facilitate an understanding of the disorders of this gland. The adenohypophysis or anterior lobe contains eosinophilic and basophilic hormone-secreting cells. These hormones function by stimulating various other glands and structures in the body. The eosinophilic cells secrete five such hormones: (1) somatotropic, which stimulates growth and protein tissue anabolism, (2) thyrotropic, which stimulates the thyroid cells to become hyperplastic and secrete thyroxin and is inhibited by thyroid hormone, (3) adrenocorticotropic which stimulates the production of hormones which affect electrolyte metabolism, protein anabolism and gluconeogenesis, (4) glycotropic which stimulates the conversion of glycogen to glucose and is therefore called diabetogenic, (5) lactogenic which initiates lactation in the female breast suitably prepared with estrogen.

The basophilic cells have been shown to secrete: (1) follicle-stimulating hormone, FSH, which stimulates the Graffian follicle to grow and produce estrogen in the female, and stimulates the seminiferous tubules in the male. It is inhibited by estradiol. (2) Luteinizing hormone, LH, which induces ovulation and the formation of a physiologically inert corpus luteum in the female, and in the male stimulates the Leydig cells to produce testosterone. It is inhibited by progesterone in the female and by testosterone in the male. (3) Luteotrophic hormone which stimulates the corpus luteum to manufacture progesterone. There is evidence that this hormone is identical with lactogenic hormone produced by the acidophilic cells.

The posterior lobe produces pitressin which induces peripheral vasoconstriction and stimulates smooth muscle. It contains an anti-diuretic principle which increases tubular reabsorption. Pitocin causes contraction of the uterus when that organ is under the effect of estrogen but not when it is responding to progesterone.

A direct measurement of anterior pituitary function may be obtained by bioassaying the amount of follicle stimulating hormone excreted in twenty-four hours (8). Other laboratory aids in the diagnosis of pituitary disorders are:

1. Skull roentgen ray—sella turcica.
2. Glucose tolerance test, insulin sensitivity test, and/or a combination of these two—abnormalities in glycotropic or adrenocorticotropic hormone production.
3. Excretion of 17 keto-steriods—adrenocorticotropic.
4. Visual fields—involvement of optic tracts by an expanding pituitary tumor.
5. Basal metabolic rate and cholesterol—thyrotropic deficiency.

Panhypopituitarism (Simmonds or Sheehan's Disease). The fundamental aberration in this infrequent disease is underproduction of all anterior pituitary hormones. Signs of thyrotropic lack (pituitary myxoedema) or signs of gonadotropic lack may predominate, but a combination of tropic deficiencies must be present.

A number of the patients suffering from this disease are women whose illness dates back to a pregnancy which was complicated by severe hemorrhage, toxemia or sepsis. In other patients, tumor, xanthoma, trauma or infection may cause the necessary atrophy or pressure necrosis of the anterior lobe.

The symptoms and signs correspond to the deficiency states produced by a lack of the various tropic hormones. Thyrotropic deficit causes torpor, cold sensitivity, and a thick tongue with slurring speech. Glycotropic deficiency is manifested by increased glucose tolerance with a pronounced tendency to hypoglycemic episodes. Weakness, low blood pressure, diarrhea and nausea demonstrate the adrenocorticotropic dysfunction. Amenorrhea, loss of libido, impotence and sexual atrophy are due to gonadotropic deficiency.

The problem of differentiating this disorder from the non-endocrine disease, anorexia nervosa may be difficult. The following positive findings in the latter disease are of differential importance: frequent occurrence in frustrated spinsters; growth of a silky lanugal hair; basal metabolic rate not lower than −20 per cent (panhypopituitary cases often range from −30 per cent to −40 per cent), mental alertness, and response to psychic therapy.

Therapeutic measures for panhypopituitarism have progressed rapidly in the past ten years. Replacement therapy with desoxycorticosterone acetate, dessicated thyroid, estrogens, progesterone and testosterone may restore these patients to satisfactory mental and physical activity. The administration of thyroid without concomitant desoxycorticosterone acetate therapy may precipitate an Addisonian crisis.

Acromegaly. This disorder is usually the result of an eosinophilic adenoma of the anterior lobe, although it may be caused by simple hyperplasia of the eosinophilic cells. There is a marked overgrowth of certain body tissues with the production of the characteristic facies, large hands and feet. Frequently there is overproduction of the thyrotropic hormone, with resultant signs and symptoms of increased metabolism, and an overproduction of diabetogenic hormone, causing a mild diabetes mellitus. Atrophy of the basophilic (gonadotrophic) units with a resultant amenorrhea or impotence is frequent. Severe headache, hypertrophic arthritis and renal calculi may occur.

Extension of these tumors backward and upward to encroach upon the optic nerves, optic chiasm or hypothalamic structures produces the visual disturbances such as hemianopsias or blindness which may accompany this disease. These may appear quite suddenly, and demand surgical intervention. Otherwise treatment consists in radiation therapy, control of the hypermetabolism and carbohydrate abnormalities with endocrine substitutive therapy. With proper care, the disease is of long duration; for the tumors respond well to radiation.

Chromophobe Adenomas. Although the chromophobe cells are endocrinologically inert, adenomas arising therefrom may produce varying degrees of hormonal deficiency by pressure replacement of functioning chromophilic tissue. The symptoms vary considerably and a knowledge of the basic manifestations of underproduction of the various "tropic" hormones permits prediction of the signs and symptoms. In addition, pressure on the optic chiasm and hypothalamus may account for other changes. The primary defects are:

1. Hypogonadism—secondary to gonadotropic lack and characterized by amenorrhea, genital atrophy and azospermia.
2. Hypothyroidism—secondary to thyrotropic lack but without the dry, scaly skin and the bloated facies. Instead the hair is fine and the skin

smooth and soft. The basal metabolic rate may drop to very low levels (circa −40 per cent).

3. Hypoglycemia—secondary to glycotropic deficiency.
4. Optic chiasm pressure—hemianopsia, bitemporal or homonymous is often followed by blindness. The tumor may invade the floor of the third ventricle in a degree sufficient to cause internal hydrocephalus.

The signs of a chromophobe adenoma which has penetrated the sellar roof to involve ocular or hypothalamic functions dictate surgical removal of the accesible portion of the tumor. Here, radiation therapy is usually unsuccessful in contrast to the good results anticipated with eosinophilic adenomas. Substitution therapy with desoxycorticosterone, dessicated thyroid, and estrogens or androgens plus dietary regimes for the often associated obesity may be beneficial.

In a considerable number of patients dying from Cushing's Syndrome, basophilic adenomas are demonstrable in the adenohypophysis. However, present day concepts propose adrenal cortical hyperfunction as the true causative mechanism (9), and accordingly the condition will be discussed under this heading.

Anterior pituitary hypofunction prior to puberty produces the aspects of pituitary dwarfism or infantilism. Anterior pituitary hyperfunction before ossification is complete produces gigantism, and accordingly these disorders will be summarized in a subsequent article on Endocrine Disorders of Childhood.

Dystrophia Adiposa Genitalis (Fröhlich's Syndrome). This rare condition is characterized essentially by obesity, sexual infantilism and at times by dwarfing. It is caused by a lesion involving the anterior lobe, the posterior lobe and/or the hypothalamus. Diabetes insipidus is a frequent accompaniment and narcolepsy an occasional one. Pressure on the optic tracts produces hemianopsias often associated with optic atrophy, and the diagnosis should be questioned in the absence of visual changes. The fingers are slender, small and tapering, and the bodily habitus is decidedly feminine. A subnormal basal metabolic rate and increased sugar tolerance are often demonstrable.

It should be emphasized that obesity and small fingers do not warrant a diagnosis of this rare disease. The demonstration of true sexual infantilism, visual disturbances or diabetes insipidus and one or more of the other changes listed is absolutely necessary before the fat person with a feminine habitus is diagnosed as having pituitary disease.

Diabetes Insipidus. The posterior lobe of the pituitary gland is the terminus of fibers which arise in the supra-optic nuclei, sweep backwards through the floor of the third ventricle to traverse the hypothalamus and ramify in the posterior lobe. This functional unit is termed the supraoptico-hypophyseal or the hypothalamo-hypophyseal tract. Any disorder affecting the functional integrity of this tract may result in a diminution or absence of the anti-diuretic hormone with resultant polyuria, polydypsia and weight loss. The urine shows a specific gravity below 1.008; there is hemoconcentration and hyperchloremia.

A number of diseases have been reported to cause diabetes insipidus. Cerebral and pituitary neoplasm, xanthomatosis, lymphoma, syphilis and other diseases may be so situated as to interrupt the continuity of the supraoptico-hypophyseal tract. The disease must always be differentiated from so-called hysterical polyuria. In diabetes insipidus, the polyuria persists when fluids are withheld, and there is a constantly low urinary specific gravity. It is interesting that Von Hann (10) has demonstrated a diuretic action of the anterior lobe by the failure of animals to develop diabetes insipidus after total hypophysectomy. This diuretic action may be a function of thyrotropic hormone.

Three commercially available extracts from the posterior lobe have antidiuretic properties: pitressin, dessicated posterior pituitary powder, and pitressin tannate in oil. Pitressin may be used for temporary control when the other substances are not available. The powder is administered by nasal insufflation several times a day depending upon the severity of the disease. Pitressin tannate in oil is gaining in popularity, and one injection in amounts up to 1 cc. in twenty-four hours will produce satisfactory control of the diuresis.

Adrenals. The adrenal cortex secretes an indefinite number of hormones whose functions are dependent upon their chemical structure. Basically they are steroidal compounds which vary according to the radicals substituted on the various carbon atoms. Their essential physiologic effects are to stimulate salt and water retention by the body and to stimulate the conversion of amino acids to glucose (neoglucogenesis). The absence of a hydroxyl group at the seventeenth carbon atom in the steroid nucleus characterizes the compound desoxycorticosterone acetate which is necessary for life by reason of its control over salt and water metabolism. The carbohydrate regulating hormones have an oxygen atom at C 11 and raise the blood sugar by stimulating neoglucogenesis. An androgenic hormone which stimulates the growth of axillary and pubic hair is also manufactured in the adrenal cortex.

The androgenic hormone of the adrenal cortex and the androgenic hormone of the testicle are excreted in the urine as steroidal compounds with a ketone group on the seventeenth carbon atom and a free methylene group. Accordingly, they are called neutral 17-keto-steroids, and by use of the Zimmerman color reaction it is possible to determine the amount of these substances excreted in the urine in twenty-four hours. Thus in the female, the excretion is a direct measure of adrenal cortical function, and in normal adult females, 9–10 mgms. are excreted in twenty-four hours. In normal adult males, another 5 mgms. of testicular androgenic derivatives are excreted, bringing the normal male level up to 14–15 mgms. The level is influenced by absorption from the gastro-intestinal tract, liver function, nutrition, adrenal cortical function and pituitary function. Thus it may be low in anorexia nervosa, liver disease, malnutrition, myxoedema, Addison's Disease and panhypopituitarism. In the latter two diseases, the level may reach zero, while in adrenal cortical hyperfunction as a result of hyperplasia or tumors the level is often high (10).

Hypoadrenocorticism (Addison's Disease). In about 40 per cent of the cases this disease is caused by tuberculosis (11), while in the remainder, varying degrees of simple atrophy of the adrenal gland are noted.

The basic aberrations in this disease are as follows: As a result of lack of salt controlling hormone (desoxycorticosterone acetate), there occurs an increased excretion of sodium and chloride associated with retention of urea, potassium, and phosphorus. Profound dehydration with low blood sodium and chloride levels ensue. The following signs and symptoms appear: weight loss, weakness, anorexia, nausea, vomiting, hypotension, abdominal pain and diarrhea, the last being variable. There is to a lesser degree a lack of the carbohydrate producing hormone with a resultant low blood sugar, a subnormal temperature and a tendency to spontaneous hypoglycemia. Probably as a result of androgenic deficiency, amenorrhea, loss of libido, impotence and frequent loss of axillary and pubic hair appear.

The most striking manifestation of this disease is a brown, grey or blue pigmentation with melanin which is prone to occur on mucosal surfaces such as buccal, rectal or vaginal, and on scars, creases and pressure sites. In his original paper, Addison (12) noted the additional occurence of areas of vitelligo and leukiderma. The diagnosis of Addison's Disease is extremely doubtful in the absence of pigmentation, and the deposits are not materially affected by therapy.

In addition to the biochemical abnormalities listed above, various other laboratory procedures are of diagnostic value. The neutral 17-keto-steroid excretion is low to absent and the basal metabolic rate is below 0 per cent. Chest roentgen rays reveal a small heart and may demonstrate tuberculosis. Glucose tolerance tests may accentuate a latent hypoglycemia, and there is usually a low white blood count with a relative lymphocytosis.

If the patients with Addison's disease remain unrecognized, either in the natural course of the disease or as the result of a complicating infection or operation, the individual may develop with amazing rapidity clinical aspects characterized by a sudden intensification of all signs and symptoms, associated with a profound peripheral vascular collapse. These may be precipitated in treated patients by a complication as seemingly minor as an acute sore throat, and it represents basically a state of acute severe salt and water deficiency. It is called Addisonian crisis. In 1938, Cutter, Power and Wilder (13) outlined a test for Addison's disease based on the deliberate withdrawal of sodium chloride from the diet as a means of intensifying salt and water loss. To be sure, this procedure is frequently diagnostic, but it may precipitate a true adrenal crisis, and it is therefore not recommended for clinical use.

More recently, Robinson, Power and Kepler (14, 15) have devised a "Water Test" which is based on the inability of these patients to exhibit a spontaneous diuresis when given a large amount of water. If this procedure is positive, the blood and urine are analyzed for urea and chloride. The patient with Addison's disease will show urea retention and increased chloride excretion. A positive result may also be obtained in pituitary insufficiency, liver disease, hyperthyroidism, anorexia nervosa and in patients who have had gastric resection (15).

The use of the glucose tolerance test as a diagnostic aid in patients suspected of Addison's disease or panhypopituitarism may precipitate profound hypoglycemia. Following an initial rise, the blood sugar often falls to low levels from which it will not return without the administration of intravenous glucose. This phenomenon which Albright (16) calls hypoglycemic unresponsiveness is probably caused by rapid diversion of glucose from the blood stream in an attempt to replenish depleted glycogen stores.

The treatment of Addison's disease is the administration of sodium chloride and adrenal cortical hormones, and it has been placed on a sound footing largely through the work of Thorn and his associates (11, 17). A small group of patients may be maintained on therapy with sodium chloride alone. The majority require desoxycorticosterone acetate, and the most convenient method of administration is by pellet implantation. The hormone requirement is first determined by administering daily injections of desoxycorticosterone acetate, the average patient requiring less than 5 mgms. per day. In addition, the patient is given from 3–8 grains of sodium chloride per day, and the response is measured by daily weight gain, relief from symptoms, rising blood pressure and correction of biochemical abnormalities. Signs of overdosage are hypertension and edema; at times the patients may complain of severe joint pain. When optimum dosage has been obtained, pellets are implanted into the infrascapular area after calculating the number required. Standardized pellets (Ciba: 125 mgms. each) are absorbed at the rate of 0.3–0.5 mgm. per day, and therefore have a life span in the body of approximately two hundred and fifty days. Although theoretically one pellet should be implanted for each 0.5 mgms. of hormone required, it is far safer to implant one or two pellets less than the number so calculated, covering any deficit by administering added salt by mouth. In this way, overdosage necessitating removal of pellets will be averted. Since adrenal steroids with carbohydrate regulating potency are unavailable for clinical use, and an inordinate amount of cortical extract would be required, the treatment of hypoglycemia consists in the administration of a high carbohydrate diet with frequent feedings. The use of testosterone proprionate to increase protein anabolism has been recommended by some workers (18). In questionable cases, cautious hormone and salt therapy with careful observation of the patient for prompt increase in weight, strength and blood pressure is indicated.

Hyperadrenocorticism. An understanding of the syndromes falling under this heading will probably be aided by certain general statements. Excessive production of adrenal cortical hormones produces signs and symptoms which may be divided into metabolic and sexual changes. The clinical disorder predominated by metabolic changes is termed Cushing's syndrome, and that predominated by sexual changes is termed the adrenogenital syndrome. There is no constant purity to this delineation, and a varying number of manifestations characteristic of the one syndrome will appear in the other.

In 1932, Cushing (19) called attention to the frequent association of buffalo obesity, hypertension, glycosuria, etc. and basophil adenomas of the pituitary gland. He believed that this syndrome which now bears his name was caused by the pituitary basophilism. This concept brought forth a storm of criticism which centered about the facts that basophilic adenomas may occur in patients who show no evidence of Cushing's syndrome (20, 21), and Cushing's syndrome may occur without basophilic adenomas (22). The present concept is that Cushing's syndrome is caused by hyperfunction of the adrenal cortex which may occur as an idiopathic entity, as a result of hyperplasia or tumors of the adrenal cortex, or as a secondary manifestation of pituitary basophilic adenomas. Those cases associated with hyperfunction of the anterior pituitary are termed Cushing's disease, and all other cases are termed Cushing's syndrome. Albright believes that a majority of the manifestations of this disorder are the result of increased production of gluconeogenetic hormone with resultant protein depletion caused by rapid conversion of amino acids to glucose (9).

The characteristic appearance of an individual affected with this disorder is easily recognized. The manifestations include: painful "buffalo-type" obesity, hypertension, purplish-red striae on the abdomen and legs, a plethoric moon face, acne vulgaris, hirsutism and osteoporosis. The patients have a thin skin and easy bruisability; amenorrhea and loss of libido are other accompaniments.

Despite the importance of adrenal cortical hyperfunction in the production of this syndrome, characteristic changes in blood electrolytes are relatively infrequent. In 45 cases collected from the literature, Soffer (10) found an elevation of blood sodium in 4 instances, a reduction of blood chloride in 5 instances, and a reduction of blood potassium in 8 instances. The excretion of urinary 17-keto-steroids will usually be elevated in those patients with hyperplasia or tumor of the adrenal cortex, the highest values being obtained in the latter group. In adrenal cortical carcinoma, Talbot et al. (23) have demonstrated that there is an increase in the urinary excretion of the beta fraction of the keto-steroids. In addition, those patients with malignant adrenal cortical tumors will usually show excessive daily excretion of urinary androgens and estrogens.

A majority of the patients will show decreased tolerance on glucose tolerance tests, and a few will exhibit manifest diabetes which is difficult to control. Hematologic studies will reveal a mild polycythemia in a minority of the cases.

Roentgen ray studies are invaluable in the diagnostic sorting out of these cases and should include investigation of the sella turcica for signs of pituitary tumor and visualization of the vertebrae and long bones for osteoporosis. Adrenal enlargement may at times be demonstrated by intravenous pyelography and peri-renal air insufflation.

In discussing this disorder, mention should be made of other entities which may enter into the differential diagnosis. Knies and Le Fever (24) have reviewed a series of cases occuring usually in females and characterized by obesity, hirsutism, disturbed menses and signs and symptoms of diabetes mellitus. There are symmetric hyperostoses on the inner table of the frontal and parietal bones, and the disorder is termed *hyperostosis frontalis interna*. The cause is unknown. In addition, certain malignant tumors of the thymus are associated with changes characteristic of Cushing's syndrome, and these changes are probably caused by an associated adrenal cortical hyperfunction.

The treatment of Cushing's syndrome should probably proceed as follows. A period of administration of testosterone proprionate in conjunction with a high protein intake in an attempt to replace depleted protein stores. This is followed by adrenal exploration with the removal of the tumor or the removal of one gland if bilateral adrenal hyperplasia is present. The immediate pre-operative treatment should consist of the administration of large amounts of cortical extract and salt. The post-operative mortality is high in those cases with tumor, and a picture which resembles an acute Addisonian crisis may supervene.

Adrenogenital Syndrome. These cases are characterized by varying degrees of aberration in sexual structure and function as a result of increased production of androgenic hormone. Metabolic changes characteristic of

Cushing's syndrome may occur simultaneously but are not of particular diagnostic aid.

The pathology varies according to the age of the patient and ranges from an absence of any demonstrable adrenal pathology to large adenocarcinomas.

In general, the manifestations of this disorder are pseudohermaphroditism, precocious sexual and physical development, hirsutism, amenorrhea, impotence, a change in voice and a tendency to sex reversal.

The congenital form of the disease is characterized by pseudohermaphroditism. True hermaphroditism implies the presence of testes and ovaries, and is an embryologic developmental defect not related to adrenal cortical pathology. Pseudohermaphroditism is characterized by the presence of gonads of only one sex associated with abnormalities of the external genitalia which make sexual identification difficult; abdominal exploration is frequently necessary for final identification. Those individuals with testes are designated male pseudohermaphrodites, and there is often no demonstrable adrenal cortical pathology. In contrast, those cases with ovaries, designated female pseudohermaphrodites, usually have adrenal hyperplasia or tumor.

The females usually exhibit an enlarged clitoris, hirsutism and rudimentary ovaries, tubes and uterus. Frequently the vagina opens into the urethra instead of on the exterior. Male pseudohermaphrodites usually have a varying degree of hypospadias, partially descended or pelvic testes and often a cleft scrotum which resembles a vulva.

Especially in female patients, the possibility of an adrenal cortical tumor must be borne in mind. Procedures of diagnostic value include a keto-steroid determination, pyelography and retroperitoneal air insufflation and estrogen determination. Laparotomy is often desired for both diagnosis and treatment.

The adrenogenital syndrome appearing between birth and puberty is frequently caused by malignant adrenal cortical tumors. It may be characterized by marked obesity or by excessive muscular development, the so-called infantile Hercules. Subtle metabolic changes characteristic of Cushing's syndrome may contribute to but never dominate the clinical aspects. In the female there is enlargement of the clitoris and hirsutism. In the male child the phallus and testes attain adult proportions at any early age, and in both sexes rapid growth with early epiphyseal closure occurs.

The exact role which the pituitary, thyroid and adrenal glands play in the growth process is still not clear. It is well recognized that excessive production of pituitary growth hormone, of thyroxin or androgen results in excessive soft tissue and skeletal growth; and underfunction of these glands is associated with dwarfism. Dogmatically, it may be said that after the first year of life, the growth hormone of the anterior pituitary gland is the chief growth promoting agent, and the thyroid permits or facilitates this action. The testicular androgens act as secondary growth promoting hormones and stimulate masculine muscular development in adolescent boys. These latter hormones thus stimulate skeletal growth and precipitate epiphyseal closure (26).

In view of the above facts it is clear that an excess of androgen will result in a markedly accelerated growth rate for a short period of time with premature complete cessation of growth. Accordingly, such a patient at the age of 10 years is unduly large and at the age of 18 years is unduly small.

In summary, the disease in females is characterized by hirsutism, hoarseness and masculine contours in addition to the accelerated growth rates, enlarged clitoris, and varying degrees of metabolic change. In males there is precocious sexual maturation and excessive growth with varying degrees of metabolic change.

The diagnosis and treatment is the same as outlined for the congenital form of the disease.

The adult form of this disease is quite rare. Virilism with a hoarse voice, large clitoris, masculine contours, amenorrhea and hirsutism comprise the symptom complex in females. In males, the disease presents two distinct aspects. The first group is characterized by feminization with genital atrophy, breast enlargement, marked weight fluctuation and loss of libido. The second group is characterized by obesity, loss of libido and varying degrees of metabolic change as described under Cushing's syndrome.

Diagnostic and therapeutic procedures should be pursued as described under Cushing's syndrome. Particular attention must be paid to the exclusion of ovarian arrhenoblastomas.

Here an attempt has been made to delineate hyperadrenocortical syndromes into cases in which sexual changes and those in which metabolic changes predominate. Such delineation is often difficult, and there is a constant overlapping of signs and symptoms so that it is most unusual to see "sexual syndromes" without metabolic changes and vice versa. This fact must be borne in mind at all times.

Parathyroids. The parathyroid glands are small oval structures, usually four in number, situated posterior to the thyroid gland. Their proximity to this structure renders them particularly liable to removal in the course of a subtotal thyroidectomy. Parathormone, the secretory product of these glands, is intimately concerned with the metabolism of calcium and phosphorus, and a brief recapitulation of pertinent facts regarding the physiologic variation in these ions seems indicated.

Approximately 99 per cent of the body's calcium is present in the form of a calcium-phosphate-calcium carbonate compound in the bones, teeth and body fluids. Calcium is present in the blood largely as calcium ions and calcium proteinate, the former physiologically active. Accordingly, a determination of the serum protein is indicated, in conjunction with calcium/phosphorus determinations, for a low protein level with a normal calcium level implies an increase in functionally active calcium ions. Normal blood calcium levels lie between 9.5 and 11.5 mgms./100 cc., and since this is above the threshold for calcium excretion, calcium normally appears in the urine. Calcium deposition and calcium absorption from bone are going on constantly under normal conditions. The absorption of calcium from the gastro-intestinal tract is stimulated by vitamin D, and a subnormal serum calcium level stimulates the parathyroids to produce hormone.

Phosphorus normally occurs in the blood at a concentration of 3–4 mgms./100 cc. as an inorganic phosphate. It is also found in the bones and teeth.

Probably the chief effect of parathormone is to increase the excretion of phosphorus in the urine, which lowers the serum phosphorus level. This calls forth phosphorus and calcium from the bones producing rarefaction of this tissue and a high serum calcium level. When the serum calcium is raised sufficiently, hypercalcuria occurs (26). The theory that the primary action of parathormone is to remove calcium from bone (25) has been largely supplanted by the theory outlined above.

Hypoparathyroidism. A small number of cases of chronic idiopathic hypoparathyroidism have been reported (28), but the majority follow accidental removal of some or all of these glands during thyroidectomy. The serum calcium is low, the serum phosphorus is high, and there is no calcium excreted in the urine. As a result of the low serum calcium, the classical signs of tetany develop. There is increased neuromuscular irritability with resultant numbness and tingling, carpopedal spasm, laryngeal spasm, and infrequent epileptic seizures. The historic signs of Chvostek, Trousseau and Erb are positive, and in long-standing cases, cataracts frequently occur. Development will cease in those teeth which had not completely matured before the onset of the disease. The bones are denser than normal.

Tetany is also caused by alkalosis which is most commonly caused by an emotional hyperventilation. Here the respirations are deep but not necessarily rapid, and there is obvious emotional stress. The carbon dioxide combining power of the serum may be only slightly reduced, and the attacks respond rapidly to rebreathing in a paper sack or holding the breath. Other causes of alkalosis tetany are the ingestion of large amounts of alkali and excessive loss of gastric contents. Rickets, renal insufficiency and steratorrhea are also associated with a low blood calcium.

Several compounds are available which effectively elevate the serum calcium to normal levels. Parathyroid hormone is useful in the immediate control of postoperative hypoparathyroidism and may be administered in a dose of 1–3 cc. by injection. This should be accompanied by the intravenous administration of calcium gluconate until the blood calcium reaches normal levels, or until calcium appears in the urine as detected by the Sulkowitch test in which a reagent containing oxalate buffers causes a precipitation of calcium oxalate (29). Parathormone in amounts of 0.2 to 0.4 cc. may be administered every twelve hours for several days and should then be replaced by more reliable compounds, for parathormone rapidly loses its effect (supposedly as a result of antihormone formation), is expensive and may cause local reaction.

Maintenance therapy is best obtained by the use of the compound dihydrotachysterol or A.T.10 which is an oily solution containing 5 mgms. of active principle per cc., effective by mouth. If it is desired to control the disease at its onset with this material, about 3 cc. a day should be given until the Sulkowitch test is positive. The maintenance dose is usually 1 cc. three times a week, and the patient himself can modify the dose according to the results of the Sulkowitch test.

The diet should include a high calcium and low phosphorus intake, therefore milk should be omitted for it is high in phosphorus content. Sufficient calcium may be obtained by the use of a prescription for 10 cc. of 30 per cent solution of calcium chloride diluted in water taken three times a day after meals.

Hyperparathyroidism. In 50 cases of this disorder reported by Fuller Albright, 41 were the result of single adenoma, 3 cases of two adenomas and 6 cases of hypertrophy of all of the parathyroid tissue (30). The adenomas are almost always benign. The four cardinal chemical changes around which the clinical aspect matures are hyperphosphaturia, hypophosphatemia, hypercalcuria and hypercalcemia.

The symptoms may be divided into three groups, the first of which results from the chemical alteration of the blood, i.e. hypercalcemia and hypophosphatemia. These symptoms include muscular weakness and atony, fatiguability, constipation and anorexia.

The second group of symptoms refers to the increased excretion of calcium and phosphorus in the urine which results in single or multiple renal calculi or a diffuse calcification of the renal parenchyma termed nephrocalcinosis, polyuria and polydypsia. In a recent series from the Mayo Clinic, renal calculi were reported to occur in 80 per cent of the cases (31). Cope has reported that 10 per cent of renal calculi are caused by hyperparathyroidism, but this figure is high, probably as a result of the great concentration of cases of hyperparathyroidism at his clinic (32). The stones are composed of calcium phosphate or calcium oxalate, and progressive renal insufficiency because of pyelonephritis or hydronephrosis caused in turn by renal calculi frequently ensues.

It is most important to realize that skeletal lesions are not a constant part of the aspects of hyperparathyroidism. Their occurrence is probably related to the amount of calcium in the person's diet; a high calcium intake maintaining a positive calcium balance despite hypercalcuria. When bone lesions occur, there is first a generalized decalcification of bone—osteitis fibrosa cystica generalisata. Next there occur manifestations as a result of pronounced decalcification in certain areas, notably the long bones, skull, ribs, metacarpal and metatarsal bones. These areas may consist of aggregations of osteoblasts and osteoclasts signifying pronounced local metaplasia—the benign giant cell tumor or osteoclastoma. Bone cysts and brown tumors of the jaw (epulis) are also seen with considerable frequency. However, not every epulis is the result of hyperparathyroidism.

The diagnosis may be simple, more often difficult. Accurate and frequent chemical determinations are essential, and the following aberrations are sought: (1) hypercalcemia; the calcium level fluctuates constantly and frequent determinations are necessary; (2) hypophosphatemia; a low phosphorus level is more constant than is a high calcium level; (3) hypercalcinuria. The Sulkowitch test, previously described, may be used to screen out those patients who show excessive excretion of calcium. If this is present, the patient should be placed on a diet in which calcium intake is accurately measured (33) and twenty-four hour urinary calcium determinations obtained. On this diet an excretion of over 150 mgms. per day is considered suspicious, and over 200 mgms. a day is regarded as pathognomonic. Other causes of hypercalcinuria include a high calcium intake (dairy products and nuts especially), and pathologic states as listed in the accompanying chart. Serum alkaline phosphatase determinations are elevated only in the presence of bone disease.

FIGURE 1

	Serum Ca	Serum P	Serum Alk. Phosphatase	Urine Ca	Bones
Hypoparathyroidism......	L	H	L-N	L-Absent	Thick
Rickets..................	L-N	L-N	H	L	Thin
Renal rickets............	L-N	H	N	H	Thin
Hyperparathyroidism......	H	L	H-N	H	Thin
Osteoporosis.............	N	N	N-L	H-N	Thin
Osteomalacia.............	N-L	L	H	H-L	Thin
Paget's disease..........	N	N	H	H-N	Spotty
Metastatic malignancy....	H	N	N-H	H-N	Spotty
Multiple myeloma.........	N-H	N-H	N	H-N	Spotty or Thin

H—high; L—low; N—normal.

Roentgenographs are of the utmost importance and should include dental views as an effort to detect a disappearance of the lamina dura, a calcium line which normally invests each tooth. Attention to the roentgen ray detection of renal calculi or nephrocalcinosis is indicated.

The differential diagnosis includes a long list of conditions characterized by variations in calcium phosphorus metabolism, and for simplicity a differential chart is appended (see Fig. 1). The picture of hyperparathyroidism with extensive renal damage and phosphorus retention producing a high serum phosphorus level may present a serious problem. Hyperplasia of the parathyroid glands with apparent compensatory increase in function is encountered in chronic renal insufficiency, rickets, osteomalacia, multiple myeloma, and some cases of carcinomatosis (34). In children, this produces renal rickets and in adults, renal osteitis fibrosa cystica; both characterized by generalized decalcification.

Renal rickets is associated with certain features which are of aid in differentiating it from hyperparathyroidism. (1) There is a high serum phosphorus level and a high alkaline phosphatase. (2) There is chronic acidosis as a result of persistent loss of base. (3) There is a history of preceding renal disease.

The only satisfactory means of treating hyperparathyroidism is by removal of the hyper-functioning parathyroid tissue. Cope has well described the difficulties inherent in this procedure (35, 36). A diet low in calcium should be prescribed until operation, and in those patients with severe bone disease, the post-operative course is frequently complicated by tetany.

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A NEWER TREATMENT OF SCABIES*†

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Prior to reports on the use of benzyl benzoate in 1912 by Nielson and in 1937 by Kissmeyer (1) on the treatment of scabies in man, various preparations of sulfur, betanaphthol, balsam of Peru, tar, and styrax were used. Sulfur was the most reliable and widely used drug among this group, and still occupies a prominent place in the therapy of scabies. All of these drugs caused dermatitis in a large number of the patients treated. When, however, scabies is complicated by secondary infection with pyogenic organisms, the therapeutic problem presented concerns not only the eradication of the animal parasite but also the treatment of the pyogenic dermatitis as well. All of the aforementioned drugs are active scabiecides, but none of them are effective in the treatment of purulent cutaneous dermatoses; therefore, the additional use of some other preparation is indicated.

Kissmeyer (1) in his report on the treatment of 8000 cases of scabies with a single application of a benzyl benzoate emulsion claimed uniformly good results. Slepyan (2) and Graham (3) made similar claims. Mellanby, et al. (4) demonstrated that a 25 per cent emulsion of benzyl benzoate will kill the acarus scabiei on a glass slide in five minutes. Carpenter, et al. (5) stated that therapy with benzyl benzoate was well tolerated by their patients and that this form of therapy caused fewer reactions than sulfur when used in any form. They also claimed that their treatment time was greatly reduced by the use of this drug. Goldman (6) recommended the use of a benzyl benzoate cream and claimed good results in all of his cases with a maximum of four to five applications. No reactions were encountered in his series. Lutz (7) claimed excellent results and no recurrences. Brain (8) states that in the treatment of scabies in children a 25 per cent emulsion of benzyl benzoate is the drug of choice. If secondary infection is present, he recommends that the scabies be treated first and the purulent infection handled as a separate problem later. Dobes and Nippert (9) treated one series of patients with sulfur and a second series with benzyl benzoate. In the second series they encountered no reactions. There have been a few reports of reactions following the use of benzyl benzoate. Daughtry (10) reported 4 severe reactions which were manifested as scarlatiniform and reddish papular eruptions which disappeared within five to ten days after the use of the drug was discontinued. The authors have encountered several cases in which a contact dermatitis developed following the use of a benzyl

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benzoate preparation, but few of the reactions were severe and the dermatitis involuted without any sequellae when the drug was discontinued.

Tyrothricin, a substance obtained from the soil bacilli, was first used by Dubos (11) in 1939. Herrell and Heilman (12), Francis (13), Rammelkamp and Keefer (14), and others reported excellent results following the use of tyrothricin in the treatment of superficial skin infections. It has been used both as a continuous wet dressing and mixed in various ointment bases. Anderson (15) claimed good results in 75 per cent of his cases when he used wet dressings. The authors (16) found tyrothricin to be an effective agent in the treatment of pyogenic dermatoses, and in their series only 2 cases developed a complicating dermatitis following its use.

It was thought that a combination of these two drugs should prove to be effective in the treatment of scabies, especially in those cases complicated by secondary pyogenic infection.

PREPARATION USED IN STUDY*

The following formula is the one used in the conduct of this study:

Tyrothricin	0.05%
Benzyl benzoate	30.0%
Benzocaine	3.0%
23 H Alcohol (ethyl)	65.0%
Distilled water and flavoring agents q.s.	

This formula was applied in accordance with the instructions given in a following paragraph.

CASES STUDIED

Forty-four cases are included in this study. Nineteen of the patients were children whose ages ranged from 2 months to 14 years, and of these 12 were females and 7 were males. There were 25 adults of which 9 were males and 16 were females. In this group there were 22 Negro and 22 white patients. These patients include both private and clinic cases.

All the patients treated in this series were carefully examined on each clinic visit. Only those cases were used in which the characteristic vesicle could be demonstrated with its burrow. All the patients in this group presented some secondarily infected lesions, although the severity varied greatly from a few ecthymatous lesions on the buttocks in some patients to numerous crusted and pustular lesions on the trunk and extremities in others. Patients were treated until all symptoms had subsided.

METHOD OF CONDUCTING TREATMENT

Each patient was furnished with a bottle containing 6 ounces of the benzyl benzoate-tyrothricin mixture and was instructed to use it in the following manner:

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* Furnished through the courtesy of Sharp and Dohme.

1. Open blisters and remove crusts with a needle.
2. Take a warm bath with copious amounts of soap and water.
3. Dab dry.
4. Rub the solution on lightly from neck to toes twice daily for the next two successive days.
5. After the second day take a bath as at first and then report to the doctor.
6. Scabies is a contagious disease and every member of the family that is infected should be treated.
7. All bedclothes, underwear, towels, etc. that comes into contact with the infected skin should be sterilized by boiling.

RESULTS

A noteworthy result in all but one of the cases treated was the rapidity in which itching was controlled. Some of the patients with enough intelligence to evaluate their subjective symptoms reported that almost all the itching had subsided in twenty-four to forty-eight hours. The mothers of the children reported that the children slept well after the first forty-eight hours of treatment. The patients were observed every forty-eight hours for the presence of any characteristic lesions of scabies. No lesions were found after four days of treatment. The length of time necessary to heal all lesions completely varied with the severity of the case, but in no instance exceeded fourteen days. The shortest time necessary for complete involution was six days.

There was one failure in this series, and this was a private patient, who, after fourteen days of treatment still had symptoms, and it was necessary to change to a sulfur preparation for a satisfactory result.

REACTIONS

No serious reactions were encountered in this series. Some of the patients complained of a temporary feeling of burning or tingling which immediately followed the application of the mixture but which quickly subsided. The mother of the 2 month old child stated that the child cried after the treatment and that the skin became quite red. This was not observed when the child was examined; therefore the treatment was not changed and the child recovered from scabies in six days. It was necessary to discontinue the treatment in one case because of an untoward reaction.

SUMMARY

Forty-four cases of scabies, most of which were secondarily infected, were treated with a tyrothricin-benzyl benzoate mixture. No serious reactions were encountered in the series treated. A satisfactory result was obtained in all but one case.

CONCLUSIONS

1. Forty-four cases of scabies were treated with a tyrothricin-benzyl benzoate mixture.
2. A satisfactory result was obtained in all but one case.
3. One moderately severe reaction was encountered.
4. This method of treatment may be recommended in the treatment of scabies complicated by secondary coccogenous infection in children or adults and obviates the necessity for using more than one method of treatment, or more than one preparation.

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REPAIR OF CRANIAL DEFECTS BY CAST CHIP-BONE GRAFTS*†

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On March 13, 1946, S. A. W., a girl, aged 8 years, was admitted to the Union Memorial Hospital in a critical condition. She had been struck by an automobile and sustained a depressed compound fracture of the right parietal area of the skull with contusion of the brain. Immediately upon admission, debridement of the comminuted fragments with elevation of the depressed viable fragments was performed, leaving a cranial defect about 2½ inches (6.25 cm.) in diameter (Fig. 1). The wound healed satisfactorily without evidences of infection. But of her comatose condition, nutrition was maintained by parenteral and nasal feedings. On March 22, she regained consciousness and speech returned on April 3. Spastic paralysis of the left upper and lower extremities had developed and remained unaltered when she was transferred to the Kernan Hospital for Crippled Children on April 19 for further treatment.

On April 21 Dr. Winthrop Phelps examined the patient and reported: "This child shows all the evidences of a left spastic hemaplagia of Area 4 (Brodman) type". Under active physical therapy gradual use of the left arm and leg was noted, and by June 14, three months after the accident, she had fair function in the arm and began to move the leg voluntarily. However, there was an obvious herniation and pulsation of the brain (Fig. 2) whenever the child was recumbent, or cried in the upright position. In addition, she complained of violent headaches and occasionally had projectile vomiting. She was highly emotional and irritable and subject to fits of crying. At this point consideration was given to some means of repairing the osseous defect of the skull.

Restoration of the contour of cranial defects was achieved during World War I by the use of costal cartilage grafts (8), tibial osteoperiosteal grafts (6), and foreign materials such as celluloid and silver plates. A later technique employed for the filling in of relatively small defects consisted in the removal of portions of the outer table of adjacent areas of the skull and transferring them to the defective areas. More recently bone for large grafts has been taken from the ilium. However, because of the difficulty in obtaining sufficiently large grafts for extensive defects and of giving suitable curvature

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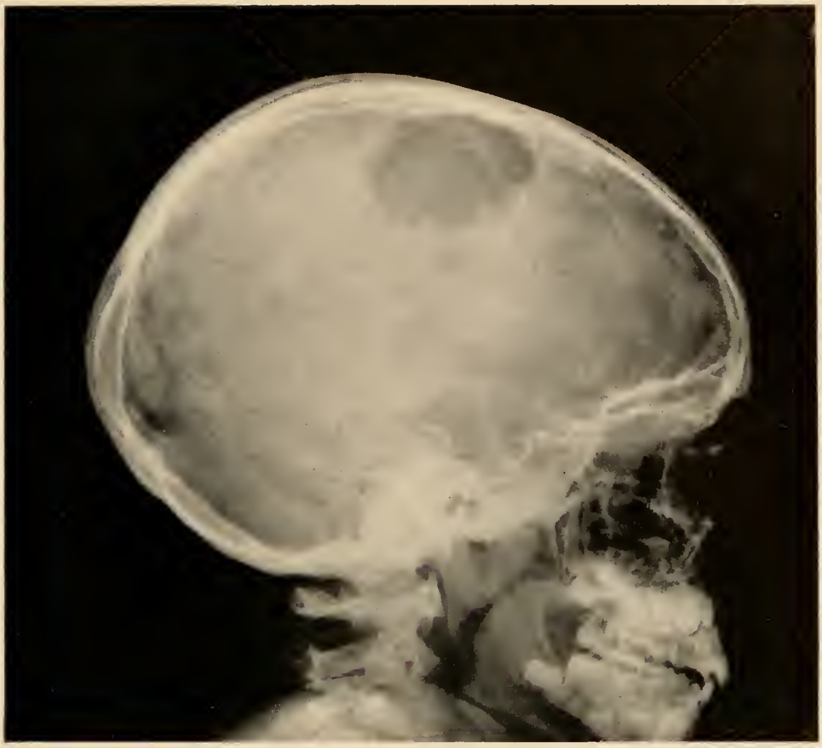

FIG. 1a. Roentgen-ray of the skull, showing the cranial defect

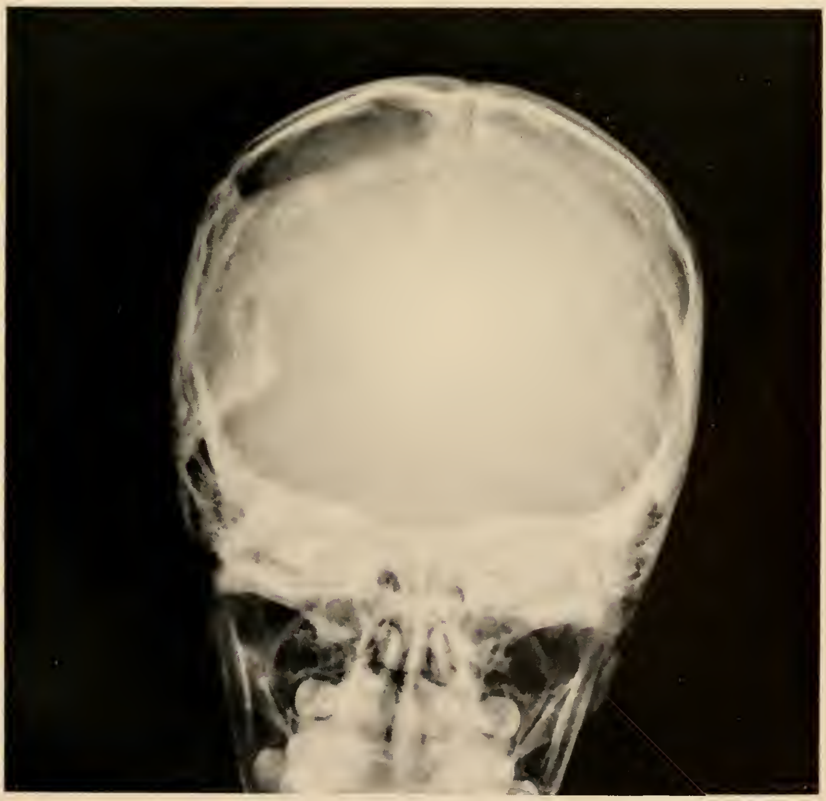

FIG. 1b. Roentgen-ray of the skull, showing the cranial defect

and shaping to the bone, multiple small cancellous and cortical iliac bone chips have been used because they can be molded more accurately to the contour of the defective area (1). Non-irritating foreign materials employed for the same purpose have been vitallium, tantalum (7), triconium, and acrylic resins. Vitallium requires preliminary casting, a serious inconvenience. Tantalum and triconium can be applied in sheets and are easy to shape and are fairly well tolerated where there is no motion, but at times slip away from their positions and leave an unsightly hump on the exposed portions of the skull; besides, they have the disadvantage of always remaining foreign bodies. The tolerance by tissues to acrylic resins has as yet not been fully ascertained. The loss of bone, however, is best replaced by bone (1) since this is physiologically and biologically logical.

One of the co-authors (C. D. C.) (3, 4) was responsible for the development of a method of using metal dies and counter dies to mold bone chips into grafts of suitable shape and thickness, for the repair of sizable cranial defects. This was first done at the French Surgery Center at Algiers during World War II. In six cases operated upon by Converse (5), and others by Campbell (2) consistently good results were obtained. As a result of this experience a suitable technique was evolved. The repairs were made by inserting a preformed mass of molded bone chips into the skull defect. This cast of bone chips when placed in the defect finally incorporates itself into the surrounding bone and ultimately restores contour and gives functional and mechanical support to the brain. It is obvious that all evidences of local infection should have been cleared three to six months before any procedure is undertaken and that the skin is stable and not unduly scarred.

The preparation of the cast-mold represents a considerable amount of exacting work before the operative procedure, but in turn the surgery following is greatly simplified. Preparation of the mold is described in detail by Clarke (3) from the making of an agar mold of the entire face of the patient, a plaster cast from this mold, plasteline and wax models of the defect and subsequently from the final model a metal die and counterdie which is used to compress the chips into the desired shape.

Satisfied that this method offered the best manner of filling in the defect in the child's skull, the dies were made, and the operation was performed on June 15, 1946, twelve weeks after the original injury.

THE SURGICAL PROCEDURE

Since this procedure is a "combined operation" performed at the same time upon two portions of the body, the skull and the ilium, it is best done by two teams. Under a suitable general anaesthetic, as the operations are begun, the patient receives intravenously blood plasma or whole blood and other necessary supportive treatment.

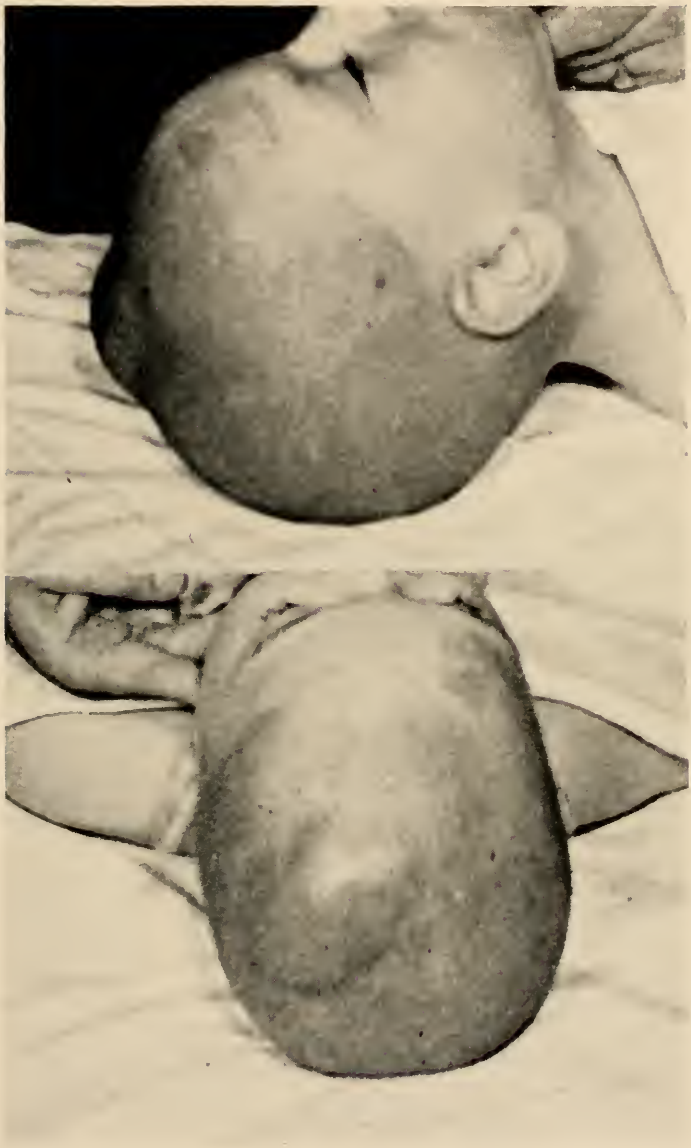

Fig. 2. Side and frontal views on the operating table, showing the hernial bulge

The iliac crest is exposed subperiosteally. Segments of bone, mainly cancellous, are removed and converted into chips no larger than one square millimeter in size; because, if they are too large, the natural elasticity of the large pieces may spring the resulting cast-chip and bone grafts out of

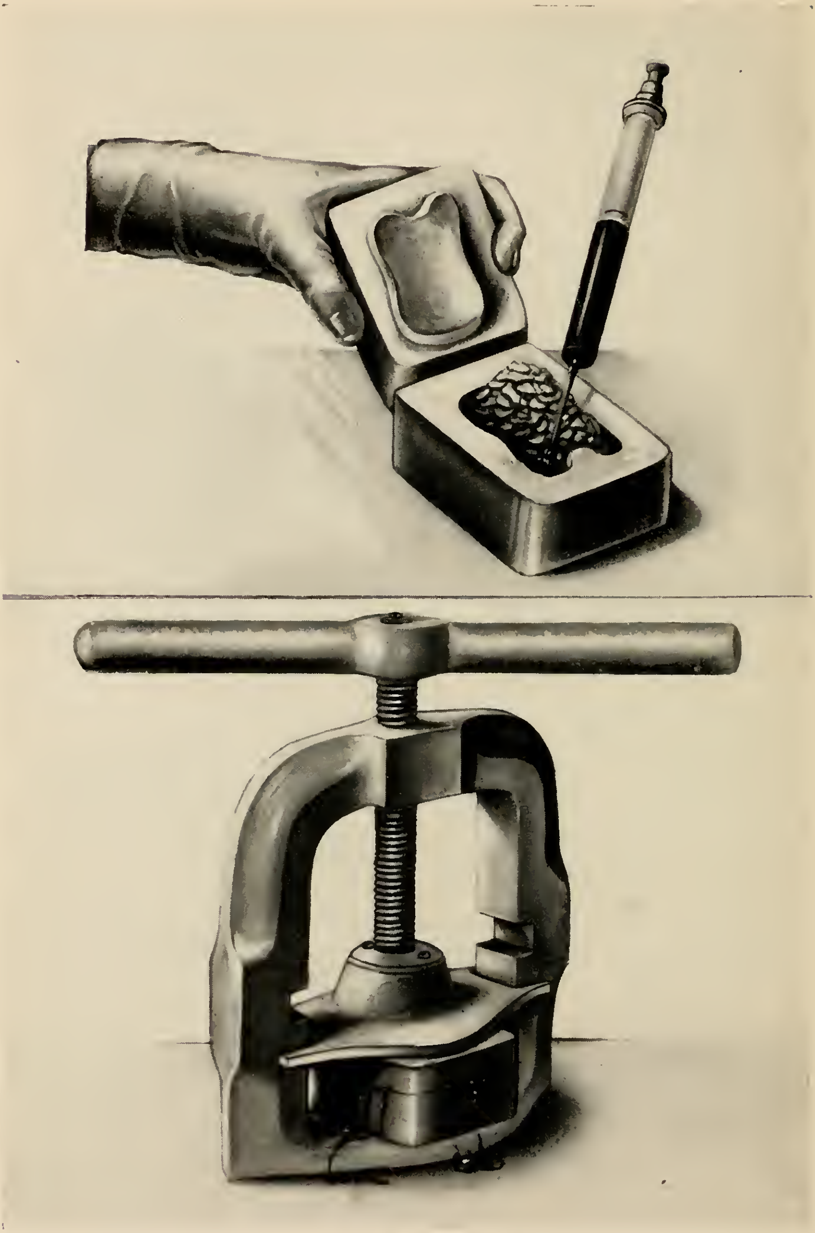

Fig. 3. Blood poured over the bone chips in the mold. The mold is placed in a dental press.

shape. Sufficient chips are taken to more than fill the lower half of the sterilized metal die and must be packed into the die to a height above that of the desired thickness of the final finished graft to insure impaction of the fragments when they have been compressed. Blood from the iliac wound is withdrawn into a syringe and poured over the bone fragments in the mold.

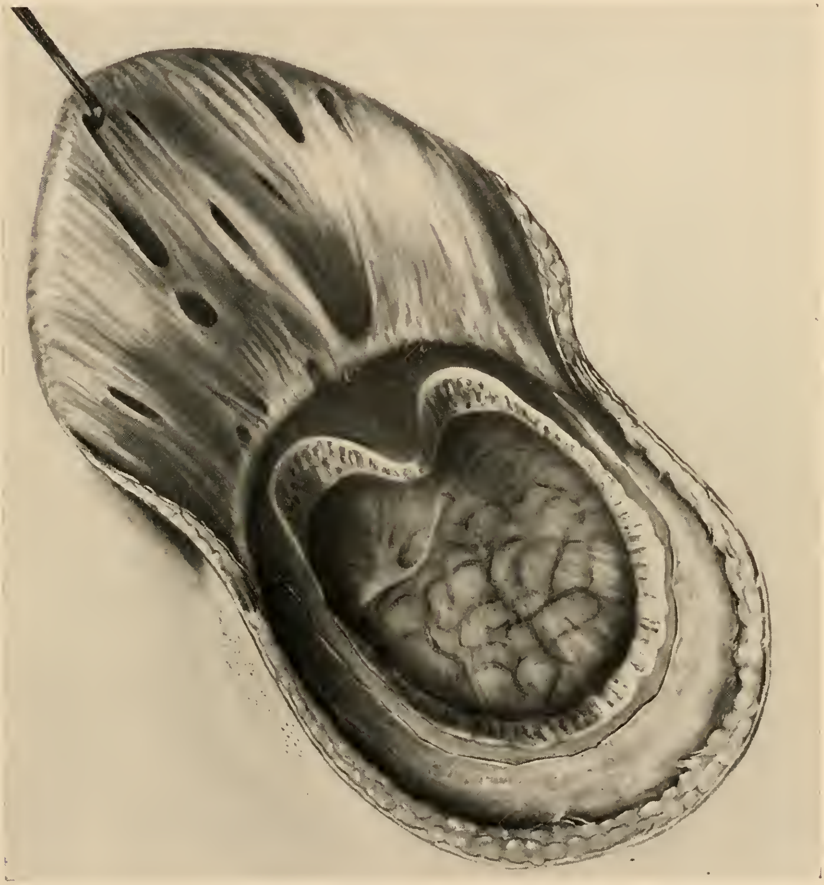

FIG. 4. Preparation of the cranial bed for reception of the preshaped bone graft

The fibrin of the developing clot acts as a cement in holding together the bone particles under pressure (Fig. 3). The upper half of the die is set in place and both halves are inserted into a sterilized strong dental or letter press and pressed together for at least ten minutes so that the cast chip bone graft will emerge as one solid mass. Failure to do this results not only in irregularities on the surface of the graft but also in flattening the curvature of the graft which would prove detrimental to the contour restoration. When the press is ready to be released this bone plastic or preshaped cast of bone is held together into one mass of proper shape, thickness and size by the double action of compression and fibrin cement. It is suggested that the dies be coated beforehand with a thin film of sterile mineral oil so that the graft can be removed from them more easily and safely.

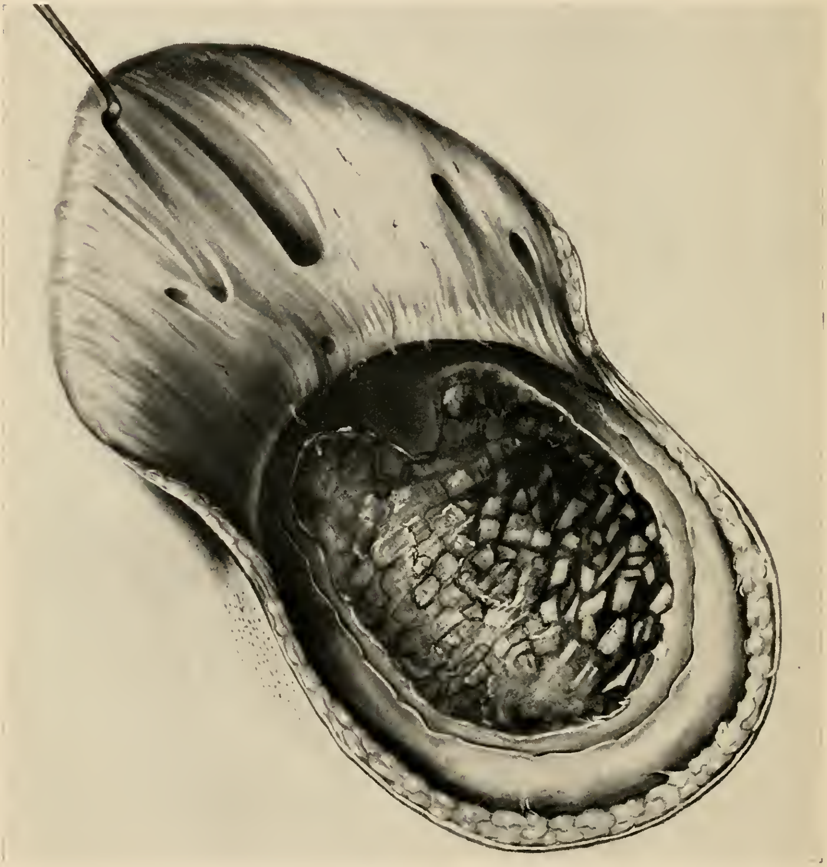

FIG. 5. The preshaped bone graft in situ

Meanwhile a suitably wide scalp flap is outlined and incised. This flap is carefully dissected from the dura, thus widely exposing the defect. The edges of the skull are freshened in a beveled manner. The defect is now ready to receive the cast chip bone graft which is being removed from the press.

The pulsation of the herniated brain makes it somewhat difficult to set the molded graft into exact relationship with the defect, but by careful compression of the brain and the scalp flap, the graft is set and held in satisfactory alignment. While the flap is being sutured, compression upon the

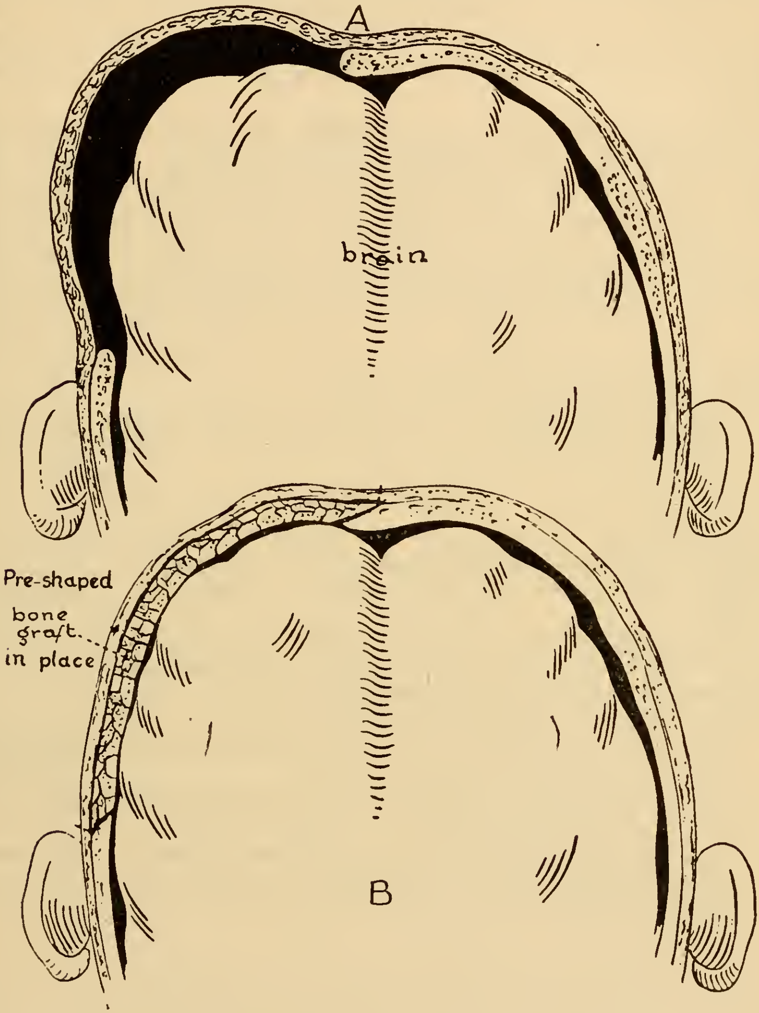

Fig. 6. A. Diagrammatic representation of condition before operation. B. After operation

graft is maintained. After closure is completed, a dressing of sterile waste is fitted snugly over the operative site and securely bandaged. An alternate method of closure is the use of a free fascial flap secured from the under surface of the scalp which is sewed to the periosteum about the rim of the crainal defect. This should steady the graft and hold it closer to the skull.

In the case of this patient, her immediate course was very stormy. She did not respond promptly to replacement or restorative therapy for more than twenty-four hours, although she regained consciousness in the usual time following the operation. Her immediate convalescence was complicated by a massive collapse of the left lung. After this cleared she gained rapidly.

By July 20, 1946, thirty-five days following the operation, the child was walking about the wards. Within six weeks the contour of the skull was fairly even and smooth. More important, however, was the fact that the cranial bulge had been entirely eliminated. No further headaches, vomiting or psychical disturbances were noted. The patient has made excellent progress in walking unaided though she still has some mild evidences of increased tonicity and spasticity in the left arm and leg. Her mental capacity has improved rapidly and her speech has cleared remarkably as have her disposition and nervous stability.

Note: The authors are grateful to Dr. Allen F. Voshell for his permission to perform this procedure on a patient on his service.

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TREATMENT OF PLACENTA PREVIA: SOME HISTORICAL AND MODERN ASPECTS*†

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While placenta previa, in the sense of the placenta presenting before the child, has been recognized as a clinical condition since the time of Hippocrates (30), its treatment has followed wide variation and even today is controversial in regard to exact methods of procedure in specific cases. It is hoped that an attempt to discern the historical backgrounds, especially with reference to certain of methods of therapy, will prove helpful in crystallizing our thoughts on the modern management of this major obstetric complication.

A brief review of the general history of placenta previa is pertinent here.

According to Fasbender (13), Deventer in 1701 coined the term "placenta previa", though his notions concerning the pathology were erroneous.

Ramsbotham (29) and Rigby (31) tell us that Paré, Guillemeau, Mauriceau, Amand, LaMotte, Dionis, and Astruc in France; Deventer in Holland; Pugh and Bracken in England; and many others, noted in their writings that the placenta may be found at the os uteri, but supposed that, "—in consequence of some peculiar accidental circumstances, it had become loosened from its attachment above, had fallen down by its own weight, and thus accidentally placed itself over the uterine orifice" (29). Another view of this matter was taken, in 1732, by Frederici (Fasbender (13)) who postulated that during labor the placenta stretched and enlarged, thus becoming separated.

It is generally agreed (14, 20, 26) that Portal, in 1664, was the first to recognize that in placenta previa, the placenta is attached to the lower uterine segment, while Schacher (Fasbender (13)) confirmed this relationship by actual anatomic dissection. Read (30), however, disagreed, stating that in Portal's works he was unable to find any reference to placenta previa. Marchetti (26) asserted that a Dutch midwife, Madam Schraders, recognized the condition independently of Portal, and realized the danger associated with it. In the middle of the eighteenth century, according to Ramsbotham (29), Giffard, Roederer, and Levret shared Portal's view, but it remained for Smellie (34) to impress the correctness of this opinion upon the profession.

Rigby is credited with being the first to differentiate clearly between placenta previa and premature separation of the placenta in 1775 (20, 22, 26, 30, 38). From reading Rigby's classic work (31), one realizes that he fully comprehended the pathology of these conditions. He also showed that in every case of placenta previa, hemorrhage must take place during cervical dilatation, and thus coined the term "unavoidable hemorrhage".

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† From the Department of Obstetrics, University of Maryland, School of Medicine.

Denman, in 1785, (Fasbender (13)) was the first to indicate the danger to the mother of gradual repeated blood loss in placenta previa.

It is of historic interest here to mention a few of the methods of treatment of placenta previa which are no longer used, such as: manual separation of the placenta before delivery, bleeding, and the administration of ergot before delivery.

Wessel (Read (30)), in 1753, recommended extraction of the placenta before delivery only when it was entirely separated and extruded into the vagina. While the method of manually removing the placenta before delivery of the child in certain cases of placenta previa is generally credited to Simpson (33), Read (30) stated that Chapman, in 1799, raised the query as to whether or not this procedure should be adopted in practice. Hirst (18) stated that this method was brought before the profession by Radford in 1819 and by Wood in 1821 as a conditional or exceptional operation to be regarded only as of occasional application. Simpson (33), just one hundred years ago (1847), advocated this method of therapy in those cases in which the hemorrhage was great and demanded immediate interference, and in which "turning" was impracticable. His maternal mortality in 141 cases thus treated was 10 or 1 in 14, while in 654 cases in which "turning" had been practiced the maternal mortality was 180 or one in 3.6. However, in the former series were included many cases in which the placenta was "thrown off" spontaneously before the child, and only 17 cases in which it was manually detached (with 1 death). Trask (38) advocated this method in 1885 in certain selected cases. However, the method seems generally to have been abandoned late in the nineteenth century.

Bleeding, years ago the sovereign remedy for many ills, also had its application and its adherents in the treatment of placenta previa. Apparently, the rationale was to let blood from the arm of the patient until the vaginal bleeding ceased. Puzos, in 1759 (Fasbender (13)) prescribed such heroic therapy in cases of severe hemorrhage from placenta previa, as had many of his predecessors, and as did many of his successors. Leroux in 1776 (Fasbender (13)) spoke strongly against blood letting in hemorrhage. Dewees (10), in 1832, was an advocate of saving the patient's blood when he wrote, "To save the woman an ounce of blood, is a duty: to save her forty, or perhaps much more, is still a greater one." However, in outlining unacceptable methods of therapy, he included, "—the lately renewed expedient of 'transfusion' ". In 1861, Read (30) stated that bleeding simply diminishes the chance of the mother's life, "—by just the amount of blood that is taken from the veins." From that time, this pernicious practice was relegated to the forgotten archives.

With the discovery by Stearns, in 1807, of the oxytocic properties of ergot, the drug was applied to the attempted solution of many obstetric problems. It is not strange, therefore, that we should find references to its use in placenta previa. Meigs (27), in 1852, recommended the administration of ergot as soon as the "turning operation" was completed. In 1855, Trask (38) advocated giving ergot to bring about uterine contractions if there were delay in their onset after rupturing the membranes. Read (30) (1861) cautioned against the use of ergot when there was a probable necessity of terminating the labor by an operation. However, the dangers of the antepartum administration of ergot were realized and the practice discontinued.

In considering some of the modes of therapy at present in use, a paragraph from Rigby (31) is quite apropos, especially when one considers the dogmatic statements of some clinics concerning their particular method or methods of treating placenta previa. He wrote, in 1784, "No circumstance that attends parturition exposes women to so much danger as profuse Haemorrhages from the Uterus towards the latter end of pregnancy, and in the time of labor; the art of midwifery is likewise, in no instance, more at a loss in the use of means for the relief of the patient; an inquiry into the causes of them and an attempt to improve the practice in such cases, cannot, therefore, be useless".

Another statement, just as true today, was made by Hicks (17), in 1885, who wrote, "The treatment of placenta previa is always a subject that it is well from time to time to discuss, in order to review the various plans suggested for its treatment under the light of increasing experience".

THE VAGINAL TAMPON

Leroux in 1776 (10, 20, 30) is reported to have been the first to recommend the vaginal tampon for any uterine bleeding, although in his writings he stated that it was recommended by the ancients. According to Holmes (20), Wigand used vaginal tamponade in placenta previa; and for over one hundred years the Rotunda Hospital at Dublin used this method almost routinely in placenta previa and premature separation of the placenta. Gardien (Dewees (10)) taught that the "plug" would do harm by stimulating the uterus, thus increasing placental separation. Trask (38) recommended the avoidance of the tampon in cases before term, and its use only for dangerous cases and those near term. Read (30) stated, in 1861, that the tampon was useful when bleeding was brisk and the cervix so rigid as to prevent artificial delivery. Hofmeier (Holmes (20)), in 1904, advocated the tampon.

Edgar (12), in 1911, believed the pack to be an efficient means of controlling hemorrhage and securing dilatation in placenta previa.

Titus (37) cautions against packing. Stander (36), in his recent textbook states that while he has never employed a tampon to the cervical canal and vagina, certain authorities recommend this procedure when the hemorrhage is alarming in cases of placenta previa with cervical dilatation insufficient to admit one finger. He does state, however, that as a temporary expedient to check hemorrhage vaginal tamponade may be used to permit transportation of the patient to a hospital. Beck (4), in 1942, listed tamponade of the vagina as a method of treatment restricted to those cases of insufficient cervical dilatation to permit other vaginal procedures.

It is with the persistent remains of this obstetric relic that we have to contend. It is believed that vaginal tamponade in placenta previa is undependable and dangerous in its action, for in most cases the pack is applied hastily and improperly with little or no antisepsis or asepsis, and therefore all cases thus treated are considered infected. In addition, the pack does not control the hemorrhage from the uterus. Holmes (20) stated that, "Its use compromises the safety of the woman when any supplemental procedure may be required, especially in the event a cesarean section is to be employed, and is particularly frought with the risk of sepsis."

SIMPLE RUPTURE OF THE MEMBRANES

"To Mauriceau, with Justine Siegemundin, 'Court Midwife to the Electorate of Brandenburg' (1690), belongs the credit for introducing the practice of puncturing the amniotic sac to arrest hemorrhage in placenta previa", (Jameson (22)). However, Read (30) stated that Puzos (1759) first performed rupture of the membranes as a substitute for the operation of version in certain cases. He further claimed that Dionis, who, with Mauriceau, is quoted by Simpson as having practiced the operation long before Puzos, does not seem entitled to this honor. Smellie (34), in 1774, and Bard (2), in 1817, recommended this method in the cases which today would be called lateral and partial placenta previa. Dewees (10) did not believe in rupture of the membranes prior to the complete dilatation of the cervix. Simpson (33) advocated artificial evacuation of the liquor amnii as good therapy in proper cases; and further stated that some authorities chose this method in complete as well as partial varieties—a trocar perforating the placenta and drawing off the amniotic fluid.

Holmes (20), Adair (1), and most other recent writers definitely accord rupture of the membranes a place in modern management of placenta previa, and it is considered the procedure of choice, when applicable, since it is effective; and the fetal and maternal morbidity and mortality are quite low.

From an analysis of the literature on placenta previa in the ten years prior to 1945, Barrett (3) concluded that the best prognosis for mother and child is found in: simple rupture of the membranes or cesarean section.

VERSION

Guillemeau (1550–1613—a pupil and son-in-law of Paré) recommended "turning on the feet" to control the loss of blood and to deliver the baby in placenta previa (Findley (14)). Trask (38) stated that Guillemeau practiced turning indiscriminately in all cases of extreme hemorrhage before delivery. Portal (1603–1703) (14), and Mauriceau (1637–1709) (15) performed podalic version and extraction by the foot in the management of placenta previa. Peu (1623–1707) (14) performed version and extraction only upon complete dilatation of the cervix, and called attention to the dangers of forcible dilatation of the cervix, especially when the patient had lost considerable blood.

Rigby (31) favored early delivery by turning, but expressed caution against performing the dilatation of the cervix and the turning too soon, since he considered this as unfavorable as performing them too late. Denman (9), in 1802, advocated immediate delivery by turning, as did Bard (2) in 1817, and Dewees (10) in 1832. The latter advised passing the hand between the placenta and the uterus so as not to pierce the placenta in performing the turning; and suggested that the version should be undertaken only when the cervix was dilated or dilatable. Ramsbotham (29) and Meigs (27) believed that version should be performed early, agreeing with Dewees that the substance of the placenta should not be broken through. Trask (38), in 1855, summarized the opinions of the day when he said, "An early delivery by turning has been sanctioned by long experience as the best general mode of treatment for securing safety to mother and child".

Read (30) stated that from 1775 to 1861, "version by the feet" was considered by the profession the most reliable mode of treating placenta previa. In 1861, he believed that turning and delivering by the feet was the main resort in most cases. He further advocated turning only in excessive exhaustion of the mother.

Internal podalic version in placenta previa is nearly always a secondary procedure following various methods of accouchement forcé. The latter are highly dangerous in placenta previa in view of the extreme vascularity of the cervix and lower uterine segment which predisposes to extensive tears and even to rupture of the uterus.

In 1860, Hicks (Hirst (18)) presented his method of bipolar version and stated that it was particularly applicable to the treatment of placenta previa. This differed from the plan of dilatation, internal version and extraction then in vogue, by combining external and internal podalic version without entering the uterus except with several fingers, bringing down a foot, and leaving the remainder of the delivery to nature in most cases.

Stander (36) stated that if the child is not viable, the Hicks' maneuver gives good maternal results; and that if hospital facilities are not available, the Hicks' bipolar version will give better results no matter what the condition of the child. Beck (4) believes that, "Unquestionably, the best vaginal method of handling placenta previa is by the use of bipolar or Braxton Hicks version".

As opposed to these views, Adair (1) says of the Hicks' version that because of its being technically difficult, deep anesthesia being required, infection frequent, serious blood loss may occur, and the baby is almost invariably sacrificed, it is limited to cases in which the baby is previable or dead where rapid control of bleeding is necessary. The strongest modern critic of the Hicks' version is Greenhill (16), who states that he is firmly opposed to its use in placenta previa, believing that it should be given up entirely, and hopes that it, "—will soon be relegated to the group of ancient operations which once were useful".

HYDROSTATIC BAG

In 1851, vanBraun invented the hydrostatic bag (8). According to Holmes (20), Barnes popularized bag therapy in placenta previa. Read (30), in 1861, said of the bag as a substitute for the ordinary modes of applying the tampon, "A more effectual mode of controlling the hemorrhage from the ruptured vessels of the os uteri cannot well be imagined, and in the cases where it has been made use of, its success was certain and prompt." Edgar (12), in 1911, described the use of various forms of bags in 10 of 40 cases of placenta previa; while Hirst (19), in 1918 advised the use of the bag, in multiparae with placenta previa, followed by bipolar version.

Titus (37), mentions the restricted use of the bag in the treatment of placenta previa. Seeley (32), in 1945, reporting a study of 250 cases of placenta previa, said of the bag (which was used in 105 of the cases), "The insertion of the dilatable bag gives good results in cases adapted to its use". Beck (4) believes that the use of the bag should be limited, "—to those cases in which only a small part of the os is covered by the placenta or in which a version cannot be done". Stander (36) claimed almost ideal results in the treatment of marginal or partial varieties of placenta previa, if the cervix is sufficiently dilated to permit of the introduction of two fingers, by the intra-ovular insertion of a large hydrostatic bag.

Holmes (20), Adair (1), and DeLee and Greenhill (8) mention some of the dangers inherent in the use of the hydrostatic bag in the treatment of placenta previa: there is an increase in the likelihood of infection which is serious in placenta previa; the presenting part is invariably displaced so that there is an increased incidence of cord prolapse and malpresentation; severe hemorrhage may recurr when the bag passes into the vagina, thus necessitating immediate major operative intervention with all the possible attendant dangers of severe trauma to the vascular lower uterine segment and cervix. DeLee and Greenhill (8) state that it is only because the hydrostatic bag is still used frequently in some clinics, that they give space in their textbook to its use.

WILLETT FORCEPS

In 1925, Willett (39) first described his fetal scalp clamp. It has been only in relatively recent years that this method has been accepted in certain clinics as a means of treating some cases of marginal and partial placenta previa. Holmes (20) mentioned the scalp clamp but stated that it frequently pulls off, while Titus (37) believed that it is superior to the hydrostatic bag. Adair (1) claimed that it is simple, and that the restricted manipulation involved reduces trauma and infection. He further believed that, "The increasing use of Willett's method should help improve the results in the treatment of placenta previa".

DeLee and Greenhill (8) advocate the use of the Willett clamp in certain cases of placenta previa. King and Chun (24) support the use of Willett's forceps in those cases of placenta previa in which cesarean section is contraindicated and artificial rupture of the membranes fails to control hemorrhage.

CESAREAN SECTION

According to Young (44), Lawson Tait was the first to suggest cesarean section in placenta previa in 1890. However, he did not perform the opertion until 1898 when he used the Tait-Porro operation. Ford, in 1892, was the first in the United States (44) to suggest this method of treatment of placenta previa. The first successful conservative cesarean section for placenta previa was performed by Bernays in 1894 (44). McPherson's paper, in 1913, was the first on this subject to receive any support, and from the date of its appearance, cesarean section came, in the United States, to have a definite place in the treatment of placenta previa (44). Tweedy, in Great Britain in 1920, advocated cesarean section as a routine method of treatment (44).

Early in the present century, the advocates of cesarean section in the treatment of placenta previa met with a storm of protest in the published writings of the members of the American Gynecological Society. Murray (28), in 1902, believed that the operation was not justifiable in the ordinary cases of placenta previa. Williams (40, 41), in 1902 and 1911, inveighed against its use as follows: "The propriety of Cesarean section for placenta previa is one which is, to my mind, very much overestimated"; "—the operation is only exceptionally indicated;" "—Cesarean section for practical purposes can be almost entirely dispensed with."

Of abdominal delivery in placenta previa, Cragin (7) in 1911 said, "In regard to the question of placenta previa being an indication for Cesarean section, the writer can only state that he has never met with the case in which he has considered it indicated."

Hirst (19), in 1918, stated that, "I have treated almost all my cases of placenta previa by Cesarean section without a maternal death —." In 1933, Cooke (6) said of this mode of intervention, "The writer, in spite of his normally very conservative tendencies, has come to believe that when the hemorrhage becomes severe, low Cesarean section should be performed by a competent abdominal operator." While Holmes (20) objected to the low section for placenta previa, he believed that abdominal delivery was the, "—ideal means of succoring the woman afflicted with a previa—if all conditions permitting the operation and definite indications are present".

Adair (1) believed that the low cesarean section is the treatment of choice in all serious cases of this condition. He further commented, "Cesarean section has steadily gained in popularity and today it can be considered the most important method of dealing with this complication."

Titus (37) prefers the low segment operation when section is indicated; while Solomons (35) stated emphatically that with certain exceptions, including lateral placenta previa, cesarean section is the treatment of choice. Barrett (3) is a strong advocate of cesarean section in most cases of placenta previa. The judicious use of cesarean section in the hands of Williamson and Greely (42) has resulted in a substantial reduction in fetal and maternal mortality. In 59 of 250 cases of placenta previa, cesarean section was used by Seeley (32), who concluded that, "In properly selected cases, cesarean section can be used to the advantage of both mother and child."

Yepes and Eastman (43) stated that, "During the past fifteen years the treatment of placenta previa has swung decidedly from vaginal methods to cesarean section; and the wisdom of the change would seem to be substantiated by numerous reports showing that the maternal mortality of this complication has been reduced to but 1 or 2 per cent when abdominal delivery is liberally employed."

"WATCHFUL EXPECTANCY"

Until recently, most writers on the subject of placenta previa believed that treatment should be instituted as soon as the diagnosis was made.

Rigby (31), Denman (9), and Bard (2), all believed that the patient's safety generally depended upon a speedy delivery and insisted upon the propriety of this procedure as soon as the "state of the parts" would permit. Holmes (20), in 1933, deplored procrastination with the following statement, "The occurrence of a first hemorrhage or a repetition of bleeding, however slight, should determine immediate agressive action". In 1940, Adair (1) stated that, "Expectant treatment is almost never indicated in placenta previa". Beck (4), in 1942, was even more positive when he said, "Active interference is indicated as soon as the diagnosis is made. There is no place for expectancy in the treatment of placenta previa." Another advocate of immediate treatment of placenta previa was Titus (37), in 1945, who said, "Pregnancy should be terminated as soon as a diagnosis of placenta previa has been established." Stander (36), in his latest textbook, stated that, "On account of the danger of profuse and unexpected hemorrhage, pregnancy or labor, as the case may be, should be terminated in the most conservative manner as soon as possible after a positive diagnosis has been made." In 1945, Barrett (3) emphasized that, "There is no expectant treatment of placenta previa."

As opposed, in some cases, to following the majority in insisting upon immediate treatment, some authors advocated a modified waiting policy. Dewees (10), in 1832, believed that before full term, when the flooding was moderate, an expectant policy should be pursued with the patient in a horizontal position. Ramsbotham (29) (1847) advised against vaginal examination before term as it might cause bleeding. Even in the face of repeated episodes of bleeding in small amount occuring before term, he advised waiting, with the patient at complete bed rest, and the avoidance of stimulation. He also instructed the patient to call instantly if the bleeding recurred. Trask (38) (1855), from a survey of all the then published cases of placenta previa concluded that, "Hence, if it be possible, cases in which premature delivery is threatened ought to be conducted to the full period."

In 1945, Williamson and Greely (42) advocated a modified waiting policy when they stated that the judicious use of cesarean section, free use of blood transfusion, and discarding the old obstetric tradition of the necessity for immediate delivery in placenta previa, have resulted in an immediate and substantial reduction in fetal and maternal mortality. In cases at or near term, they delayed treatment for about 4 days during which time the patient was gotten into the best possible condition and additional blood obtained. A sterile pelvic examination, with complete preparations for vaginal or abdominal delivery having been made in advance, was performed in the operating room on the fourth day. They further pointed out that patients may be observed for a longer period of time if the viability of the fetus was questionable and there was no bleeding.

However, it remained for Johnson (23), and Macafee (25), working independently, to focus our attention upon a regimen of watchful expectancy in the treatment of placenta previa which has already begun to revolutionize our ideas on the subject. Their principal thesis is that the initial and subsequent hemorrhages in placenta previa are rarely, if ever, fatal in the absence of rectal or vaginal examinations provided the hemoglobin is normal at the onset of the hemorrhage.

Johnson (23) reported 79 cases with one maternal death and a 22.2 per cent fetal mortality in his series treated under careful observation including an expectant policy in those cases in which the fetus had not yet reached probable viability. He emphasized that the mother's blood picture should be followed carefully, and believed this factor should determine interference. Since he believed that only about 33 per cent of cases of bleeding in the last trimester of pregnancy were due to placenta previa, Johnson (23) stated that roentgen ray examinations should be used in an attempt to determine the placental location. He further warned against any rectal examinations and vaginal examinations except in the operating room with all necessary preparations for any mode of delivery having been made. In his treatment of placenta previa, he advocated cesarean section in all cases of the central variety, and in all varieties of placenta previa in primigravidae, and rupture of the membranes in multiparae with marginal and partial types, concluding that bags, version, and scalp clamps were contraindicated in his opinion.

Macafee (25) claimed that the waiting policy reduces fetal mortality by carrying the pregnancy to as near term as possible. Vaginal examination is postponed until near term, then performed only in the operating room with all preparations for cesarean section at hand. In his hands, there has been nothing to confirm the belief that the recurrent hemorrhages tend to lead up to a catastrophic hemorrhage. Primigravidae under observation for suspected placenta previa are not necessarily kept in the hospital, provided they live within reasonable distance from the hospital, and have been warned not to permit vaginal examination; this does not apply to multigravidae. In 174 cases Macafee (25) had one maternal death and a gross fetal mortality of 23.5 per cent. He preserves an open mind with regard to the appropriate treatment of a particular case and treats it in accordance with the conditions found.

Of the above two papers Eastman (11) says they "—are progressive contributions to clinical obstetrics of the first order and will make obstetricians everywhere ponder the wisdom of their past management of placenta previa. There had heretofore been rather general agreement that placenta previa demands prompt delivery and that temporizing for any purpose is rarely, if ever, indicated. Johnson and Macafee—not only show that a waiting policy is justified in many cases of placenta previa because it improves the fetal prognosis markedly, but they also demonstrate very convincingly that such a program can be carried out with complete safety to the mother provided certain precautions are exercised."

"The recommendations in these two papers will do much good provided they do not result in callousness to hemorrhage. Certainly, they should dispel forever the old notion that a patient with placenta previa who bleeds substantially at home should be packed vaginally before being transferred to the hospital. This procedure is of no value save to conceal bleeding. The observations of Johnson and Macafee now show that it is unnecessary. The findings of these authors also demonstrate that in every case of placenta previa there is ample time to provide for blood transfusions and to make careful preparation for whatever type of treatment may seem best."

A RECOMMENDED REGIMEN FOR THE TREATMENT OF PLACENTA PREVIA

Patients who bleed in the third trimester of pregnancy should be hospitalized immediately without examination and without vaginal tamponade. Upon admission careful physical examination and blood studies should be made and the blood type and Rh factor determined. An adequate quantity of blood should be matched and held ready. Depending upon the results of the blood studies, blood should be given or not before diagnosis and definitive treatment are determined upon. Rectal examinations are interdicted.

When the above studies are completed, soft-tissue roentgen examinations should be made (the so-called placentography) in an attempt to visualize the placenta and thus determine its location. Roentgen ray films should also be examined carefully so as to detect the presence of bony abnormalities of the fetus in view of the increased incidence of gross anomalies in cases of placenta previa.

Before deciding upon the mode of treatment in any case, certain factors must be considered:

1. The general condition of the patient especially with reference to an estimation of the amount of hemorrhage and whether it is continuing; blood pressure; whether or not labor has begun; and the presence or absence of infection—actual or potential.
2. The condition of the fetus as well as its maturity.
3. The history of the patient with regard to age, parity, previous obstetric history, (size of other babies; length of labors; cesarean sections; and dystocia); whether or not there have been rectal or vaginal examinations or insertion of packs before admission; and the details concerning the onset of the symptoms preceding admission.
4. The presence or absence of other obstetric or medical complications: e.g.—contracted pelvis.
5. Differential diagnosis to exclude such lesions of the cervix as erosion, polyps, varices, and malignancy; premature separation of a low implanted placenta; and premature separation of a normally implanted placenta with external hemorrhage.
6. Determination of the type of placenta previa, which is to be done only

when it has been decided that treatment is necessary. A sterile vaginal examination should be made in the operating room where, under the so-called "double set-up", all equipment for the control of hemorrhage and for vaginal or abdominal delivery is at hand.

Having obtained all necessary information as outlined above, it is recommended that the watchful expectancy policy of Johnson (23) and Macafee (25) be followed, where possible, in all cases before term. In the cases at term, definitive treatment is to be carried out when the patient's condition will permit. Sterile pelvic examination, under the "double set-up", should be performed with the greatest gentleness in order to prevent further separation of the placenta if it is encountered. This examination will also establish such a diagnosis or detect one of the conditions which clinically may simulate placenta previa.

Active treatment recommended in cases of placenta previa (when the patient is in the best possible condition) is as follows:

1. Abdominal delivery (laparotrachelotomy) in:
    a. All cases of central placenta previa regardless of parity.
    b. All primigravidae not in labor, regardless of the type of placenta previa.
    c. All multigravidae not in labor, with partial placenta previa.
    d. All patients with cephalo-pelvic disproportion or any other obstetric indication for abdominal delivery.
2. Vaginal delivery
    a. Simple rupture of membranes in:
        (1) Multigravidae in labor with some dilatation and effacement of the cervix, with partial or lateral placenta previa.
        (2) Multigravidae not in labor with lateral placenta previa.
        (3) Primigravidae in labor with some dilatation and effacement of the cervix, with lateral placenta previa.
    b. Application of Willett's forceps in any of the cases in 2. above if simple rupture of the membranes fails to control bleeding and/or induce labor, or when it is desired to expedite delivery.

It must be emphasized that no inflexible rules should be followed and that each case must be treated according to its merits.

3. Management of the third stage of labor following vaginal delivery.
    a. Credé expression of the placenta is the most desirable.
    b. Manual removal of the placenta with great caution if Credé maneuver fails or bleeding is persistent.
    (Investigate lower uterine segment and cervix after manual removal.)
    c. Uterine, cervical, and vaginal packing if hemorrhage persists after the removal of the placenta.

4. Liberal use of whole blood before, during, and after delivery as indicated, as well as general supportive measures.

In conclusion, it is recommended that vaginal tamponade, the hydrostatic bag, internal podalic version, and the Hicks' version be entirely eliminated in the modern management of placenta previa.

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A CASE OF FATAL CORONARY THROMBOSIS IN A SUBJECT WITH EXTENSIVE PERICARDIAL ADHESIONS*†

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The present report concerns a cardio-pericardial adhesion which was observed in the cadaver of an aged male individual. This spontaneously occurring adhesion bore a certain resemblance to those which have been produced surgically for the relief of myocardial ischemia. Since the cause of death was listed as coronary thrombosis, a condition thought to be allayed by pericardial adhesions, the case seemed to be of sufficient interest to warrant a description of the condition and a study of the patient's history.

HISTORY

The subject appeared to have been well developed physically for his age. He was 84 years old, 63 inches tall, and weighed 122 pounds at death. Prior to his illness he had been employed as a dock laborer and had done heavy manual labor all his life.

The following information was obtained from hospital records. The subject was found unconscious on February 1, 1935, and was admitted to the Baltimore City Hospital. His chief complaint was a pain of two weeks duration over the precordium. The rapid heart rate was slowed by administration of digitalis, but the precordial pain persisted. The heart was greatly enlarged to the left; there was auricular fibrillation and an extra systole every fourth or fifth beat. A friction rub could not be heard with certainty. The physician diagnosed a probable coronary thrombosis or possible pericardial effusion. The patient was discharged on the 23rd day. He was readmitted on March 4, 1935, again with the complaint of precordial pain. This time there was a rough sound at systole suggesting a friction rub. He was discharged improved on March 21, but against advice. The diagnosis was coronary thrombosis secondary to arteriosclerosis. The third admission was on April 8, 1935. An electrocardiogram showed auricular fibrillation, dropped beats and severe, but localized, myocardial disease. The possibility of a coronary occlusion was considered. The final admission was on June 26, 1935, and the patient remained in the hospital until his death on October 25, 1943. During his last years the

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patient experienced frequent attacks of precordial pain, usually requiring morphine for alleviation. Death was attributed to coronary thrombosis, probable coronary occlusion and generalized arteriosclerosis. No autopsy was performed.

DESCRIPTION OF THE ADHESION

The pericardial adhesion was first noted by students who, upon opening the pericardial cavity in this cadaver, found the heart to be adherent to the diaphragmatic portion of the parietal pericardium. The examination showed an extensive adhesion of the diaphragmatic surface of the heart to the base of the parietal pericardial sac.

Figure 1 represents the heart as seen after the opening and reflection of the ventral portion of the pericardial sac. At the periphery of the adherent area, the visceral pericardium may be seen as it is reflected from the surface of the myocardium onto the fibrous sac as parietal serous pericardium. Through this glistening serous membrane, along the line of its reflection, was seen a quantity of adipose and areolar tissue which filled in the space between the myocardium and the subjacent fibrous pericardium around the margin of the area of adhesion.

At irregular intervals there appeared thickened folds of visceral pericardium which swept down from the surface of the heart onto the parietal pericardium. One of these folds was especially prominent and this band 'anchored" the heart at its apex. Although the margins of the adherent area were filled in by loosely organized areolar tissue, the rest of the field was closely fused. In this latter portion, sharp dissection was required in order to release the heart from the fibrous pericardium beneath, indicating the intimate character of the adhesion.

POSSIBLE RELATIONSHIP OF THE CONDITION TO THE ATTAINMENT OF ADVANCED AGE

In cases of coronary thrombosis the prognosis is almost invariably a poor one, and the immediate mortality is very high. Hochrein (cited by O'Shaughnessy et al., 1) reported upon a series of 297 cases in which 108 patients died in the first eight days and 48 others during the first month. It is true, however, that some patients can resume activity on a reduced level or even return to a normally active life.

In cases of recovery from coronary thrombosis there evidently occurs a vascular adjustment of some sort. Some authors have attributed the recovery to the presence of a copious intercoronary anastomosis, while others feel that the opening up of a collateral circulation via extracardiac anastomoses may enable the heart to continue to function in a satisfactory manner. In addition to collateral vessels, the heart may obtain an indirect blood supply in the event of cardio-pericardial adhesions.

The probability that intercoronary anastomoses are responsible for the recovery of some patients is not great. The question of anastomoses between coronary arteries has been the subject of much debate. In the light of recent evidence submitted by Schlesinger (2) and Blumgart et al. (3, 4) it appears, however, that the coronary arteries should be considered to be functional end arteries with intercoronary communications taking

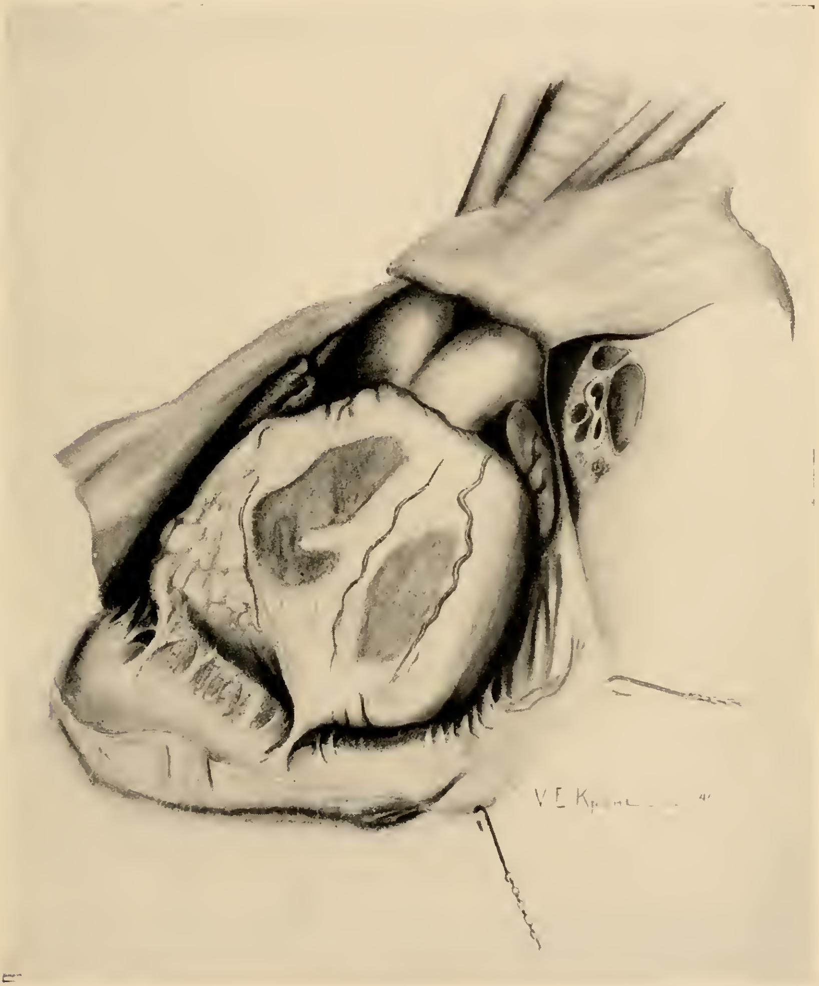

FIG. 1. The heart of an aged victim of coronary thrombosis, showing the extensive adhesions of the diaphragmatic surface of the heart to the base of the parietal pericardial sac.

place only through capillaries or tiny arterioles. There is some support for the view that extracardiac anastomoses can maintain the heart in cases of coronary occlusion, but this appears to be true only when the condition is of very gradual onset. However, pericardial adhesions have been shown to be able to prevent fatal myocardial ischemia. In experiments designed to test this contention, it was found that animals were able to tolerate drastic reductions in coronary blood supply if the heart had previously been vascularized through cardiopericardial adhesions. Beck et al. (5) have reported complete recovery of a dog with extensive pericardial adhesions in which the cross-sectional area of the coronary arteries had been reduced by about 85 per cent. The work of Thompson (6) and Heinbecker and Barton (7) may also be mentioned in which pericardial adhesions protected the hearts of dogs from myocardial infarction upon ligation of coronary vessels.

In the case which we report the coronary arteries had undergone considerable sclerosis and their lumina were greatly reduced, yet this man had reached the age of 84 years. It is of interest, therefore, to speculate upon the possible relationship of the condition seen in his heart to the age attained by the subject in the face of coronary disease of long standing (over eight years).

The adhesion between the heart and pericardium was of an extremely intimate nature, making available an additional vascular supply to the myocardium. Although it was not possible to ascertain, even appromimately, the time at which the adhesion had formed, it is certain that the conditions leading to the formation of the adhesion must have existed well in advance of the death of the patient. There was no evidence of a current inflammatory or exudative process and the visceral pericardium and serous parietal pericardium had become continuous around the margin of the fused area.

It appears quite likely that the collateral blood supply offered by the pericardial adhesion was able to mitigate the effect of either progressive myocardial ischemia or acute myocardial ischemia resulting from coronary narrowing. With his myocardium thus "protected" from coronary occlusion and infarct formation the subject was able to attain an advanced age before the occurrence of a coronary thrombosis severe enough to cause death. It would appear from this hypothesis that spontaneously occurring pericardial adhesions (i.e., without surgical intervention) constitute a natural means of off-setting the untoward effects of coronary thrombosis.

The assumption that the longevity of this individual was made possible by an indirect blood supply through the cardio-pericardial adhesion receives considerable support from the literature. One finds reports upon individuals who, although exhibiting no signs of cardiac disease during life, have come to autopsy with varying degrees of coronary occlusion, but with well developed pericardial adhesions. Thorel (8), for example, has described such a case with complete occlusion of both major coronary arteries in which the adhesions had apparently carried a blood supply sufficient to sustain the heart. Anrep (9) (cited by O'Shaughnessy et al.) has mentioned an autopsy on an Egyptian who had been accustomed to hard manual labor in spite of coronary disease of considerable duration. The occlusion of his left coronary artery and narrowing of the right coronary artery had apparently been compensated for by an adhesive pericarditis with the development of pericardial vessels. In a recent communication Cobb (10) described a rather unusual type of pericardial adhesion, which, although not as extensive as the one which we report, bears a certain resemblance to it. Cobb's adhesion also occurred in an aged male individual (71 years) and consisted of a broad serous covered band extending from the apex of the heart to the mediastinal pericardium. It was concluded that this band probably resulted from an old pericardial adhesion which had become stretched and expanded by cardiac contraction, leaving the flat apical band.

SURGICAL PRODUCTION OF PERICARDIAL ADHESIONS

During the past decade considerable interest has been aroused among surgeons concerning the production of an extrinsic collateral circulation to the heart by surgical means. The work has been based upon the theory that by supplying an extrinsic vascular supply to the heart in patients with coronary disease, the myocardium will be protected against the sudden ischemia resulting from coronary occlusion, against infarct formation and irreparable damage.

The original idea for a revascularization of the myocardium experimentally must be attributed to Beck. He and his associates have done a tremendous amount of experimental work upon the establishment of a collateral circulation to the heart (5, 11, 12, 13, 14, 15). Some of the procedures have been successfully applied as a therapeutic procedure in the human subject with coronary artery disease (14, 15, 16, 17, 18). Since the first operation for revascularization in man in 1935 (16), Beck has performed many others. A number of these operations have been reported by Beck (18) and Feil and Beck (19) and appraised by Feil (20).

DISCUSSION

In attempting to explain the presence of an extensive cardio-pericardial adhesion in an aged individual with advanced coronary sclerosis, several possibilities may be considered. One must include the possibility that it was merely by chance that this individual had previously developed an inflammatory condition which lead to the production of the adhesion.

Then later, when the normal vascular supply to the myocardium was curtailed through coronary sclerosis, the auxiliary circulation from the pericardium was prepared to maintain an adequate blood supply to the heart, thus prolonging the life of the subject. On the other hand, it may be that the formation of pericardial adhesions is Nature's method of protecting the heart against a sudden or severe thrombosis. Obviously this protection is not extended to all individuals, but in those in whom there is a gradually diminishing coronary circulation, or in whom there has occurred a series of sub-fatal coronary thromboses, conditions may be established which will lead to the formation of pericardial adhesions. Although this idea is entirely without proof, it is an extremely interesting one. Careful examination at autopsy of the hearts of patients who have survived one or more coronary thromboses or who have evidenced advanced coronary sclerosis, should throw considerable light upon the problem.

Whether occurring only through coincidence in those with coronary artery disease or through some causal relationship with the condition of the coronary vessels, pericardial adhesions appear to exercise a definitely protective influence. In the case reported here, one may surmise that the subject had benefited through this protection.

This account is offered as additional evidence in favor of the concept upon which newly developed surgical methods of inducing pericardial adhesions have been developed in an attempt to prolong the life of the patient with coronary artery disease.

SUMMARY AND CONCLUSIONS

1. In the cadaver of an aged male whose cause of death was listed as passive coronary thrombosis and arteriosclerotic heart disease, a well established pericardial adhesion was observed.
2. The anatomic features of this adhesion have been described and illustrated.
3. The general sclerotic state of the coronary arteries and blood vessels in general, as well as the history of the case indicated that the disease had existed for a long period before death.
4. The hypothesis was advanced that this adhesion may have had the effect of mitigating the influence of what would otherwise have been fatal coronary thrombosis. The attainment of the age of 84 years was accordingly made possible by the development of this pericardial adhesion which may thus be regarded as Nature's method of combating coronary artery disease.
5. This observation and concept would support the conclusions of Beck and other investigators who have demonstrated that similar adhesions induced surgically have a protective or life-prolonging influence in patients who are likely to develop myocardial ischemia caused by coronary thrombosis or other factors.

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CHRONIC GASTRIC VOLVULUS*

A Case Report

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HAGERSTOWN, MARYLAND

The word *volvulus* is derived from the Latin *volvere* which means "*to turn about or to roll*". In the modern medical dictionary it is defined as "an obstruction due to a knotting and twisting of the bowel."

This disease was first mentioned by Berg in 1866 on an autopsy report. It was fifty-four years later before its roentgenologic aspects were described by Rosselet. By the middle 1930's more than a hundred cases had been reported. However, it is still an uncommon disease.

The classification of volvulus of the stomach is by no means clear. Since, because of its peculiar ligamentous attachments, the stomach cannot be moved in any direction without "a knotting and twisting" of one or several of its parts, it seems logical to assume that any displacement is, in fact, volvulus. Classification should be simply descriptive, therefore, in regard to direction and degree, (the part involved and the symptomatology) and whether the volvulus is acute or chronic, traumatic or developmental. The one exception to the above is a true congenital anomaly of the stomach in which displacement has occurred during the early formation of the organ, and the tissues and parts have adjusted themselves to their abnormal position. Here there is neither kinking, torsion nor obstruction and hence no volvulus.

Following Singleton's classification, the case being presented may be described as a chronic (permanent) partial, anterior rotation, organo-axial, idiopathic gastric volvulus.

REPORT OF A CASE

I. B. is a white, married female, age thirty. She is of average build and weight. There is, however, a marked scoliosis involving the dorsal and lumbar spine, which according to the history has been present since a fall down stairs at the age of 9. The patient compensates for this deformity very well and carries herself in an almost normal manner. The only other significant feature of her past history is a subtotal thyroidectomy seven years ago. However, the symptoms of a mild thyrotoxicosis are still present. There have been no abdominal operations.

For three months prior to examination the patient complained of recurrent attacks of acute epigastric pain. These attacks lasted from ten minutes to several hours and occurred as often as twice a day. There has been some nausea but no vomiting. The attacks have

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* Received for publication August 12, 1947.

no special relation to the intake of food, but there has always been a persistent sense of fullness following meals. There has been no constipation.

Roentgen-ray Examination: Fluoroscopy was begun in the upright position, and a preliminary check of the chest and abdomen showed no evidence of disease or abnormality, except the previously-mentioned marked scoliosis of the lumbodorsal spine. The barium meal was taken by the patient without difficulty and outlined the esophagus which was of normal length and caliber. As the stomach filled, it was immediately noted that the barium was "puddling" in the cardiac end. The level continued to rise as the meal was swallowed; and when the glass was empty, the puddling was still apparent. No other portion of the stomach was filled. With mild peristalsis, the meal was slowly pushed somewhat up-hill and to the patient's right through a narrowed pars intermedius, and then

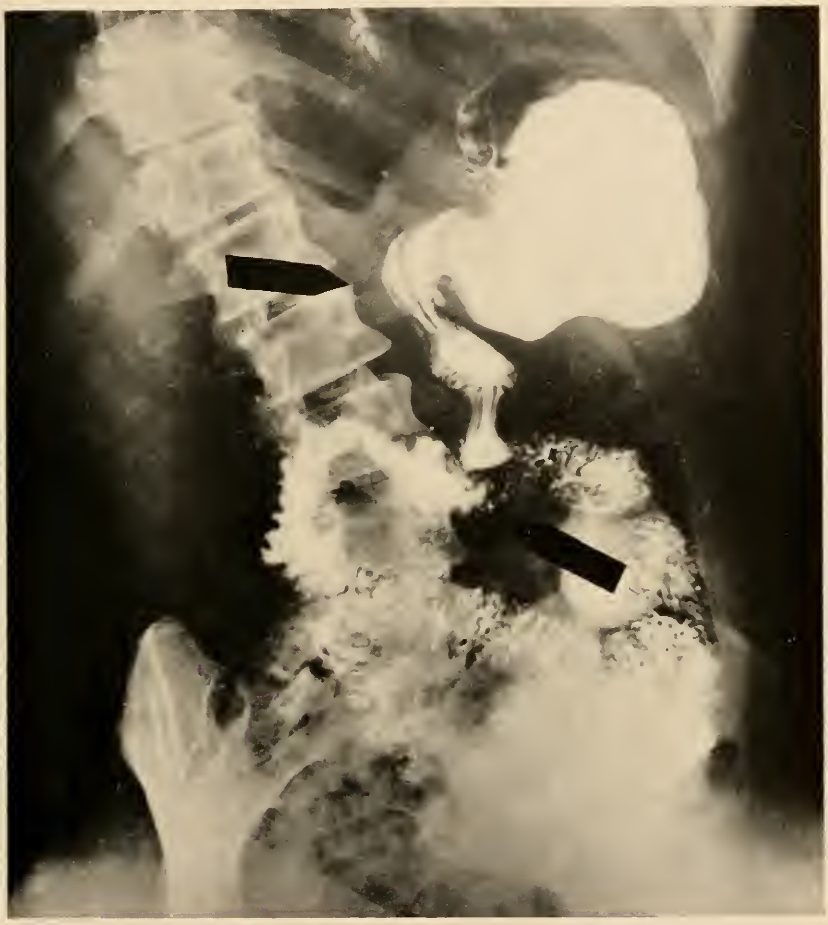

FIG. 1. Right anterior oblique projection. Note the spiral arrangement of the mucosal folds. The cap points downward and slightly to the left.

flowed rapidly downward into the pyloric antrum. The mucosal folds in this portion of the stomach were prominent and in a distinct barber pole spiral (Fig. 1). The greater curvature was cephalad. The cap filled almost immediately and its position was downward and slightly to the patient's left (Fig. 2). Its contour and action were normal. Peristalsis was not active and because of the stomach's high position palpafion was difficult. There was no tenderness. Examination of the patient in the recumbant and prone positions verified these abnormal findings. The left lateral view (Fig. 3) showed the inverted distal portion of the stomach as well as the duodenal bulb pointing straight towards the patient's feet. The small bowel filled and evacuated itself in the usual manner. At six hours the meal was distributed throughout the ascending and transverse colon. There

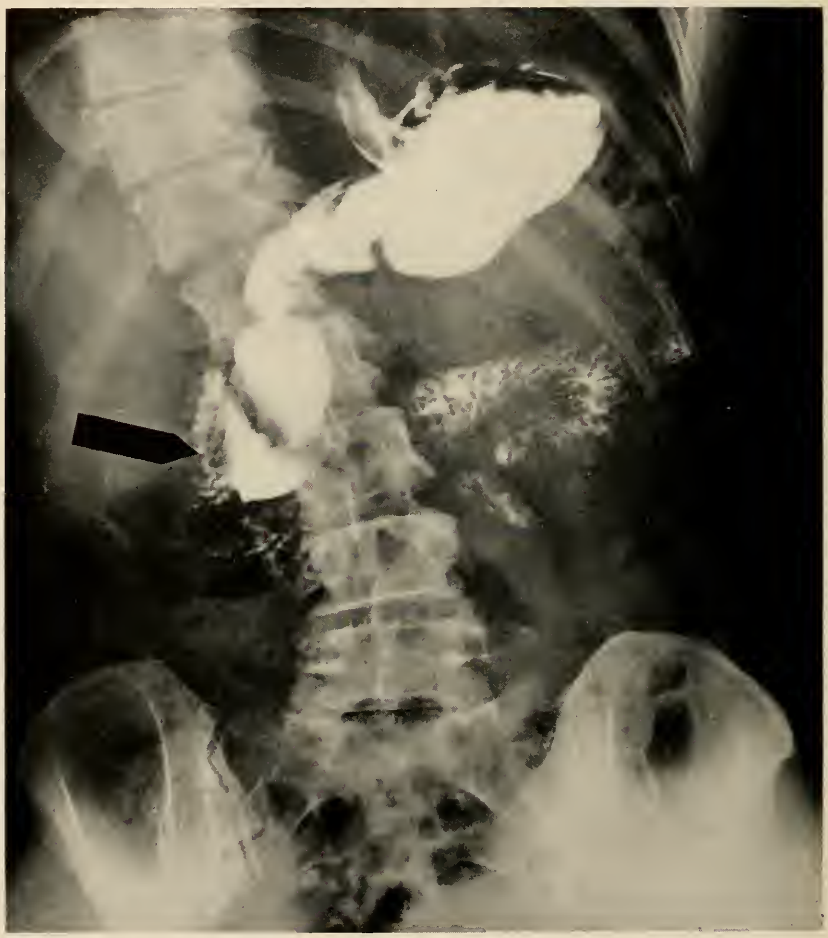

FIG. 2. P.A. projection. The greater curvature of the distal portion of the stomach is cephalad.

was no gastric residue. The transverse colon was quite high in the abdomen, particularly in its distal portion, and lay between the stomach and the anterior portion of the diaphragm.

Sequelae: Following this examination, the patient was put on a bland diet with a mild antispasmodic. She remained symptom-free and two months later was reexamined. The findings were identical. Operation is not contemplated.

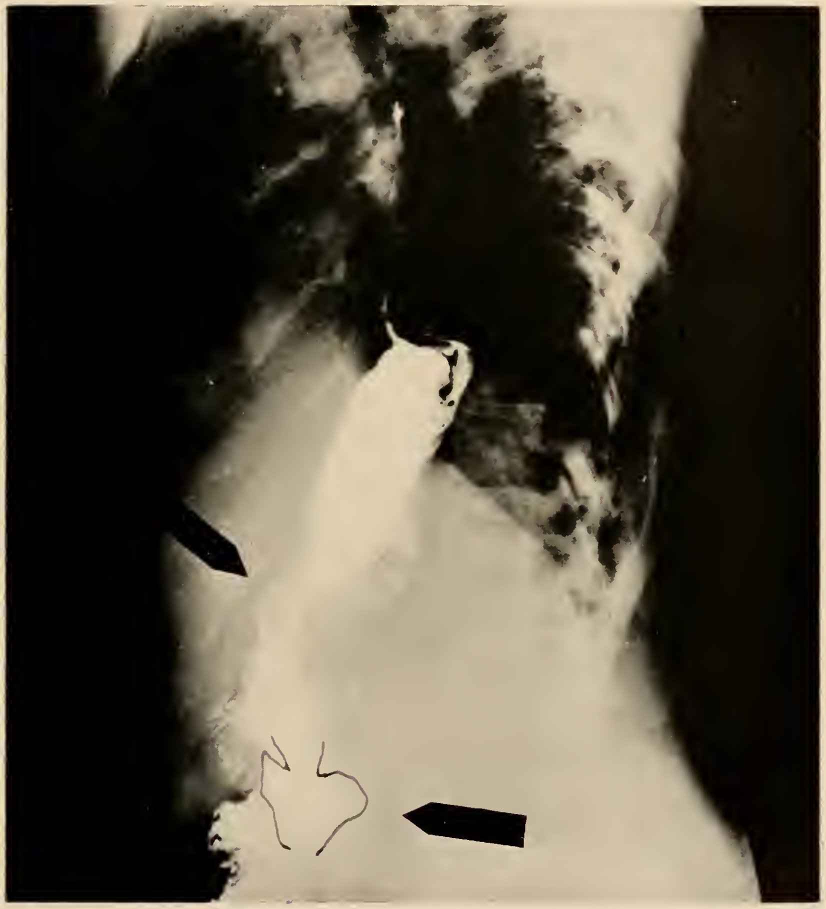

FIG. 3. Left lateral projection. The pars intermedius is twisted and narrowed. The cap points downward.

COMMENT

It is believed that this case represents a chronic permanent type of gastric volvulus with 180 degree upward anterior rotation of the distal portion of the stomach around the organoaxis (Fig. 4). This includes the distal part of the pars media, the pars pylorica, the cap and first portion of the duodenum. The normal relationship of the greater and lesser curvatures is inverted. The proximal end of the stomach is relatively fixed. The deformity of the spine is believed to be a contributing factor in the etiology.

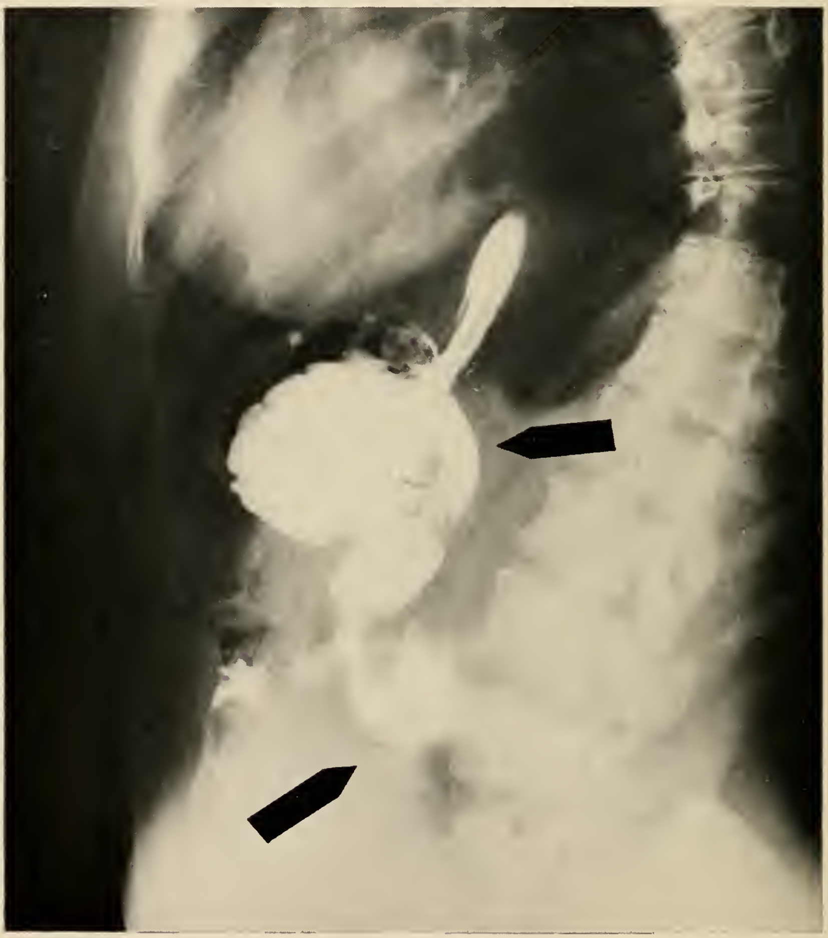

FIG. 4. Left anterior oblique projection 180 degree rotation of the distal portion of the stomach. The inverted pars intermedius and cap are designated.

This case meets the following radiologic criteria for the diagnosis of chronic volvulus:

(a) Stomach high in the abdomen with the puddling of barium under the left dome of the diaphragm. (Air in the stomach is frequently seen under both domes in this condition);

(b) Narrowed pars intermedius with the rugae in a distinct spiral;

(c) The distal portion of the stomach and the cap pointing downward and slightly to the left;

(d) The greater and lesser curvatures inverted;

(e) The transverse colon pulled up by the gastro-colic omentum to a position in front of the stomach and between it and the anterior diaphragm. The criterion of spontaneous reduction, mentioned by some writers, is not met. It is believed that the abnormality is permanent in this patient. It has probably developed over a period of years due to the deformity of her spine and the resulting relaxation of the ligamentous attachments of the stomach. A subclassification of permanent chronic gastric volvulus is, therefore, suggested.

SUMMARY

(1) A case of chronic gastric volvulus is reported and its radiologic appearance described.

(2) Some of the radiologic diagnostic criteria are mentioned.

(3) A subclassification of permanent chronic gastric volvulus is suggested.

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DEPARTMENT OF OBSTETRICS

An Obstetric Case Report*†

The patient was a 20 year old primigravida, admitted to the hospital in early, active labor at term. Her prenatal course had been uneventful. The external pelvic measurements were: Sp 24; Cr 28; Tr 32; D.B. 20. No pelvic examination had been done during pregnancy. On admission the height of the fundus was 29 cms., the fetal heart in the right lower quadrant, the presenting part dipping into the pelvis. A breech presentation was suspected. Rectal examination revealed 2 cms. dilatation with effacement of the cervix. The heart, lungs, and blood pressure were within normal limits. The patient's contractions were five minutes apart, lasting forty-five to fifty seconds. Her labor had started three hours before admission. After ten hours of labor her contractions were two to three minutes apart lasting fifty to fifty-five seconds and the cervical dilatation was 6 cms. with the presenting part, 1 cm. above the spines. At this time the membranes ruptured. Labor continued and after eighteen hours of labor the cervix was thought to be fully dilated with the presenting part level with the spines. Labor was allowed to continue, the patient remaining in good condition. After four hours of full dilation there was no progress and a consultant was called. Before the consultant arrived, her pains, which had been tumultuous, suddenly stopped. Her pulse rate jumped to 130, blood pressure 70 systolic over 40 diastolic. The fetal heart could not be heard.

A rupture of the uterus was recognized. Blood was started, and after 2 pints there was no improvement in the patient's condition. A laparotomy was then performed, the uterus opened, the baby extracted, and a large rent was seen in the left, posterior wall of the uterus extending into the broad ligament. A rapid hysterectomy was done. In 3 hours the patient received 2500 cc. of blood and 1000 cc. of plasma. However, she showed increasing signs of shock and died three hours following the operative procedure. A pelvic exmination postmortem revealed a diagonal conjugate of 9 cms. and a markedly contracted pelvis.

DISCUSSION

This case is not presented for the purpose of discussing rupture of the uterus, but to emphasize some points in prenatal care.

During pregnancy the external measurements were taken but no pelvic examination was done. External pelvimetry is unreliable, and is of no value at all unless accompanied by an adequate pelvic examination, which should include examination of the cervix by visualization, diagonal conjugate and transverse ischial. If a pelvic examination had been done, a short diagonal conjugate and contracted pelvis would have been recognized, and actually, unless there was a very small baby, elective cesarean section would have been done with probable survival of both mother and baby. External measurements are not sufficient for good prenatal care. A pelvic examination ignored is an injustice to the patient.

It is the feeling of the author that all primigravid breech presentations at

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* From the Department of Obstetrics School of Medicine, University of Maryland.

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term should have a roentgen ray cephalo-pelvimetry. One of the causes of primary breech presentation is a contracted pelvis. Even if, by pelvic examination, one feels that the pelvis is adequate for vaginal delivery, one should check the clinical findings by roentgen-ray examination.

In closing, it is wise to bear in mind that every patient, primigravida or multigravida, deserves a pelvic examination early in pregnancy (and it can be done safely up to the thirty-second to thirty-fourth week), and that every untested pelvis which is confronted by a breech presentation should have a roentgen-ray pelvimetry.

NEWS ITEMS

In April, 1947, the *North Carolina Medical Journal* brought out a William Allan Memorial Issue, which included a brief appreciation and biography of Dr. Allan by Frederick Osborn, Trustee of the Carnegie Corporation of New York, a list of publications by Dr. Allan, and an editorial concerning him.

Dr. William Allan attended Washington and Lee University, and the College of Physicians and Surgeons in Maryland. He started his practice in Charlotte, North Carolina in 1908, because it was his ambition to be a country doctor. He became Professor of Bacteriology and Parasitology at the North Carolina Medical College, and a visiting Professor of Medicine at the Medical College of Virginia. His sovereign interest, however, was in hereditary disease, and the community in which he practiced offered an ideal laboratory in which to work, because the few families there were elaborately interrelated and had lived for generations in the same locality. In connection with this work he studied genetics, and was elected director of the American Eugenics Society and later its vice president. He became director of the Association for Research in Human Heredity. In 1939, the Carnegie Corporation of New York gave him a grant with which he was able to enlarge his investigations. In Charlotte, North Carolina, he established a Family Record Office, and he became Professor of Medical Genetics at the new Bowman Gray School of Medicine in Winston Salem. He died on April 24, 1943, his great contribution to medical science still uncompleted. However, the work which he had done was a guide and pointer for medical science, which will influence its thinking and theory for many years to come.

Dr. Allan had an open, curious and pioneering mind coupled with generosity, kindliness, and affection for humanity. His death was a tragic loss for medicine.

Dr. Everett L. Bishop (U. of M. 1916) of Atlanta, Ga. has been promoted from Associate Professor of Pathology to Professor of Pathology (Neoplastic Diseases) at Emory University School of Medicine. Dr. Bishop was elected Vice-President of the International Association of Medical Museums at the recent meeting in Chicago. He has been a member of the Council since 1939, and is also a member of the Board of Governors of the College of American Pathologists.

Dr. George S. Emerson (Baltimore Medical College, 1897) has completed fifty years of practice in Fitzwilliam, New Hampshire. On June 29, 1947, an anniversary celebration was given for Dr. Emerson by the town. Besides being Fitzwilliam's only physician, Dr. Emerson has served on the school board for forty nine years. For the same period, he has been the town health officer. He is a member of county, state and national medical associations and has served as President of the Cheshire County Medical Association.

On June 19, 1947, a testimonial dinner was given at the Sheraton Belvedere Hotel in honor of Dr. Arthur Marriott Shipley to commemorate his twenty-seven years as Professor of Surgery and his forty-five years as a teacher at the School of Medicine of the University of Maryland. The toastmaster was Dr. W. Houston Toulson; greetings were given by the Honorable Thomas D'Alesandro, Jr., Mayor of Baltimore, and the speakers included Dr. H. Clifton Byrd, President of the University of Maryland, Dr. Maurice Pincoffs, Professor of Medicine, and Dr. C. Reid Edwards, Professor of Surgery.

Dr. Shipley graduated from the University of Maryland School of Medicine as the honor man in his class in 1902. He interned on Dr. Tiffany's and Dr. Martin's services and for a year was Assistant Resident in Surgery. From 1904 to 1908 he was superintendent of the University Hospital, and then did post graduate work in Germany under Dr. Chiari. During the first World War he served with the Evacuation Hospital #8 in France. In 1920 he was cited for the Distinguished Service Medal and was appointed full Professor of Surgery at the University of Maryland School of Medicine.

Dr. Shipley is a member of the American Surgical Association, the Southern Surgical Association, the Society of Clinical Surgery, the Research Society, and the American Thoracic Society. He was a member of the Board of Regents of the American College of Surgeons from 1935 to 1945 and in 1946 he was made a Governor. From 1936 to 1937 he served as President of the Medical and Chirurgical Faculty of Maryland. During World War II he served as Head of the Procurement and Assignment Service of the Third Corps Area.

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* The names listed above are officers for the term beginning July 1, 1947 and ending June 30, 1948.

OBITUARIES

Adams, William Carlton, East Taunton, Mass.; B.M.C., class of 1896; aged 71; died, March 29, 1947, of arteriosclerotic heart disease.

Crimmins, Philip Patrick, Brockton, Mass.; B.M.C., class of 1907; aged 62; served during World War I; died, February 27, 1947, of coronary thrombosis.

Dildine, Frank C., Port Jefferson, N. Y.; B.M.C. class of 1897; aged 79; died, February 7, 1947, of accidental monoxide gas poisoning.

Dixon, Charles Henry, St. Louis, Mo.; B.M.C., class of 1894; aged 75; died, March 9, 1947, of coronary thrombosis and carcinoma of the colon.

Dwyer, Frank, Bellingham, Wash.; P & S, class of 1913; aged 67; veteran of the Spanish-American War and World War I; died, March 30, 1947, of chronic myocarditis and arteriosclerosis.

Efird, Lester Julian, Tampa, Fla.; class of 1903; aged 68; served during World War I; died, March 21, 1947, of hypertensive cardio-vascular disease.

Evans, John Ebenezer, Wilmington, N. C.; class of 1916; aged 56; served during World War I; died, March 22, 1947, of coronary thrombosis.

Frederick, Gustav Henry, Newark, N. J.; P & S, class of 1900; aged 74; died, March 18, 1947, of chronic myocarditis, nephritis and arteriosclerosis.

Golembe, Julius, New York, N. Y.; class of 1924; aged 50; served during World War I; died, March 19, 1947, of coronary thrombosis.

Hammond, Henry M., Bergholz, O.; B.M.C., class of 1893; aged 78; died, February 9, 1947, of angina pectoris.

Hibbell, George L., Stanford, N. Y.; P & S, class of 1894; aged 75; died, March 17, 1947, of cerebral hemorrhage.

Judd, James M., Varina, N. C.; B.M.C., class of 1897; aged 76; died, March 7, 1947, of coronary occlusion.

Lassman, George Abraham, New York, N. Y.; P & S, class of 1906; aged 63; died, April 3, 1947, of heart disease.

Lawler, Daniel Henry, Hammond, Ind.; class of 1912; aged 59; served during World War I; died, March 15, 1947, of coronary embolism.

MacFarland, Howard David, Utica, N. Y.; B.M.C., class of 1910; aged 59; died, March 6, 1947, of skull fracture received when he fell from a third floor window.

Martyn, Herbert S., Cuttingsville, Vt.; B.M.C. class of 1898; aged 75; died, March 14, 1947, of carcinoma of the pylorus.

Mathias, Edward Lynch, Kansas City, Mo.; class of 1904; aged 67; served during World War I; died, February 3, 1947, of coronary thrombosis.

Maucher, Joseph V., Carrollton, Pa.; P & S, class of 1885; aged 81; died March 28, 1947, of coronary occlusion.

Nalitt, David Isaac, Bayonne, N. J.; B.M.C., class of 1901; aged 68; died, April 4, 1947, of coronary occlusion.

Patrick, Lytle Neale, Gastonia, N. C.; class of 1909; aged 63; died, February 12, 1947, of cerebral hemorrhage.

Reier, Adam William, Dundalk, Md.; class of 1916; aged 58; served during World War I; died, March 10, 1947, of coronary thrombosis.

Rich, Harry Herman, Newark, N. J.; class of 1912; aged 61; died, March 15, 1947, of uremia.

Templeton, Percy C. W., Irasburg, Vt.; B.M.C., class of 1904; aged 67; died, March 2, 1947, of acute arthritis and malnutrition.

Wehner, Edward F., Clarksburg, W. Va.; P & S, class of 1902; aged 71; died, March 3, 1947 of coronary thrombosis.

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CARCINOMA OF THE LUNG

A CHALLENGE TO EARLY DIAGNOSIS*†

O. C. BRANTIGAN, M.D.

BALTIMORE, MARYLAND

An increase in the incidence of carcinoma of the lung is frequently discussed (3, 10, 12), but the rate of occurrence probably is no greater now than formerly (2). The apparent increased incidence merely indicates a more careful observation on the part of the clinicians and the pathologists. It also indicates improvement in the methods of diagnosis. The challenge of earlier diagnosis is a serious and important one in spite of closer observation and improved methods of diagnosis. In the Department of Pathology at the School of Medicine, University of Maryland, Warner (14) reported 11 patients with carcinoma of the lung found at autopsy. The report covered 1100 autopsies prior to 1934. At the same institution Horn (8) reported 45 instances of carcinoma of the lung found at autopsy from 1934 to 1946. These 45 cases were discovered in a series of 2686 autopsies. Thus, there has been an increased incidence of 0.7 per cent. In a twelve year period from 1923 to 1934 at the University Hospital there were 62 patients with carcinoma of the lung, whereas from 1935 to 1946, the next twelve year period, the number increased to 237.

The author has had under his observation from February, 1943 to January, 1947 a total of 63 private patients with carcinoma of the lung. It is necessary to distinguish arbitrarily between private and service patients, since it is impossible to determine the number of service patients with carcinoma of the lung seen at various hospitals. In the private patient group 26 were obviously inoperable as disclosed by examination, and were

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* From the Department of Surgery, School of Medicine, University of Maryland.

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not subjected to exploratory thoracotomy. Thirty-seven in this series were subjected to operation and 17 were found inoperable because of metastasis. Only 20 of the patients or 54 percent operated upon had the lung removed. Of this group surely a certain number actually should have been considered inoperable so far as expecting a reasonably long survival period was concerned.

In an eleven year period from 1936 to 1946 Ochsner (10) observed 360 patients with carcinoma of the lung, of which 210 or 58.3 per cent were explored and 129 or 35.8 per cent had the lung removed. In a thirteen year period Rienhoff (13) reported 327 patients with carcinoma of the lung subjected to operation, and only 112 or 34 per cent of these had the lung removed. Both surgeons probably were influenced by a desire to help the patient and resected lungs that should have been considered inoperable. If there is such a high rate of inoperable carcinoma of the lung in Baltimore and in New Orleans, the high rate probably exists to a greater or lesser degree elsewhere, and thus the need for earlier diagnosis is evident. It is hoped that this article may help to meet the challenge of earlier diagnosis.

As yet the etiology of carcinoma of the lung is obscure and offers no real help in early diagnosis. The tumor occurs much less frequently in women. In the 63 patients observed by the author only 4 women were encountered. Horn (8) reported 5 women with carcinoma of the lung in a series of 45 patients. Rienhoff (13) found 6 males to one female in his series. The pathologic changes follow a usual pattern. It should be remembered that the lung buds from the primitive gut tract, and therefore all its elements have a common origin; accordingly, all carcinomas of the lung can properly be classified as bronchogenic in origin. The histologic pattern varies a great deal and has brought about a nomenclature other than bronchogenic carcinoma. There are certain distinct histologic patterns: the anaplastic, undifferentiated carcinoma, often called the oat cell carcinoma of the lung; and the differentiated or aplastic carcinoma, recognized as either a squamous cell carcinoma or an adenocarcinoma. Unfortunately, it is difficult to correlate the relationship of these distinct types with each other. It is also impossible to predict accurately which one will first invade locally or which one will first metastasize. As is generally true with carcinoma elsewhere, the undifferentiated carcinoma of the lung tends to be more malignant than the well differentiated one.

The central type of carcinoma occurring in a bronchus and the peripheral type found in the parenchyma are often given as examples of growths that produce an entirely different symptom complex. Frequently, however, this is not true. Carcinoma occurring in a bronchus and producing atelectasis will cause different symptoms than one occurring at the same location without atelectasis. When the growth occurs in the periphery of the lung, atelectasis cannot occur. Often, however, the presence or absence of infection will influence the number and severity of symptoms with or without atelectasis of the lung. Neither the histologic appearance of the neoplasm nor its location have enough infallible influence on the symptoms to make a clear-cut clinical syndrome.

There are no characteristic signs and symptoms of primary carcinoma of the lung. The symptomatology of carcinoma of the lung is extremely variable, extending from a complete absence of symptoms to complete pulmonary debility. However, if the pathologic changes are neglected, the symptoms can be grouped into a reasonably accurate symptom pattern which will aid greatly in meeting the challenge of early diagnosis. The symptom grouping is as follows:

I. Carcinoma of the lung without pulmonary symptoms
  A. Without metastasis
  B. With metastasis producing symptoms not associated with the lung
II. Carcinoma of the lung with pulmonary symptoms
  A. Patients without previous pulmonary disease
    1. Insidious onset of chronic pulmonary symptoms
    2. Sudden onset of acute pulmonary symptoms
      (a.) Pneumonia
      (b.) Lung abscess
  B. Patients with known chronic pulmonary disease of long standing

In the group of patients with carcinoma of the lung without pulmonary symptoms the outlook seems grave and impossible of solution. However, the increase in mass roentgenographic examinations of the chest (5) in industry, schools, and by public health departments offers considerable encouragement when early diagnosis is considered. It can be hoped that soon all patients admitted to hospitals everywhere, for any reason, will be given a routine roentgenogram of the chest. Mass chest roentgenography of healthy young adult males reported by Levitin (9) revealed approximately 8 per cent showing abnormality. The majority of the abnormalities were not clinically significant. Hilleboe (4) in 125,190 chest examinations found 1.3 per cent with pulmonary tuberculosis. It can be reasonably expected that the routine chest roentgenography of patients requiring hospital admission will show a higher incidence of pulmonary abnormalities. Of course, it will be necessary to identify all abnormal roentgenographic shadows thus revealed. Such studies will be of great value in solving the problem of early diagnosis in the group of patients without pulmonary symptoms and without metastasis. It will also reduce greatly the number of patients in the group who have, as their first abnormal sign, symptoms from distant metastasis.

In the group with no pulmonary symptoms and who present symptoms only from distant metastasis, the only hope of an early diagnosis rests in routine roentgenographic examinations of the chest. At present this is a most unfortunate group, since these patients are beyond surgical or medical help when the diagnosis is made.

Patients with no previous pulmonary disease who develop carcinoma of the lung with pulmonary symptoms offer the best opportunity for early diagnosis. Any patient 35 to 40 years old or older who developes a chronic pulmonary symptom of any type should be suspected of having a carcinoma of the lung, until such a diagnosis can be proved false. The thought of pulmonary carcinoma must be ever present in the mind of the physician. The expectoration of blood is always worthy of a complete study to eliminate the possibility of carcinoma of the lung; and in such a patient the prognosis must be considered grave until the cause of the bleeding is definitely determined. The classical triad of symptoms, cough, expectoration of blood, and pain in the chest, is pathognomonic of carcinoma of the lung. The typical pain is constant and deep seated in character and should be distinguished from the pain of pleurisy. Unfortunately, the triad of symptoms is often a late manifestation of carcinoma of the lung.

In many patients who have carcinoma of the lung, the pulmonary symptoms are ushered in by the sudden onset of acute pulmonay infection, either pneumonia or lung abscess. The patient who can be proved to have developed pneumonia and who responds promptly to the proper chemotherapy may still have carcinoma of the lung. Carcinoma of the lung must be strongly suspected when the older patient who has pneumonia fails to absorb the lung lesion found by physical examination and by roentgenography. In the past this condition was frequently diagnosed as unresolved pneumonia. The older patient with unresolved pneumoia must always be suspected of having carcinoma of the lung. When an older patient suddenly develops a lung abscess the possibility of diagnosing an associated carcinoma seems almost impossible unless it can be demonstrated by bronchoscopy or by bronchial secretions obtained by use of the bronchoscope. Unlike pneumonia, the lung abscess is a chronic disease, and there is no specific therapy. Should the abscess heal completely in a few weeks the presence of carcinoma can easily be eliminated. If the abscess persists as a chronic lung abscess, as it is likely to do, the presence of carcinoma will defy detection until it is too late for treatment or until it is externally drained and, of course, external drainage of the carcinoma will preclude any curative surgery.

Patients who have a well established and accurately diagnosed chronic pulmonary disease can, and often do, develop carcinoma of the lung. The chronic pulmonary disease may possibly mask the symptoms of the carcinoma until it is too late for treatment. Unfortunately, the chronic pulmonary disease will often mask or obscure the roentgenographic findings of the early carcinoma, and the true diagnosis will not be suspected until it is too late to offer the patient surgical treatment for the carcinoma. It is easy to suggest that a change in the patient's symptom complex should be carefully studied and evaluated. However, it is not so simple when the patient presents the combined disease for diagnosis. This group probably will defy early diagnosis but constant consideration of the possibility may be helpful.

The clinical course of the disease cannot be predicted by a study of the pathologic histology or by the location of the neoplasm. The symptomatology does not necessarily supply information that may prove valuable in judging the course of the disease. Since the actual time of onset of the neoplasm is never known, the duration of the untreated disease is likewise unknown. It is well to point out that roentgenographic studies of the chest indicate that ocasionally a patient may show a normal roentgenogram of the chest and yet have an inoperable tumor in four months. Contrary to such rapidity in the growth of the neoplasm, it is not uncommon for a growth to remain relatively stationary for as long as thirteen months, as shown by roentgenographic examinations of the chest. If, perchance, the patient is being treated for pulmonary tuberculosis because of a mistake in diagnosis, he may even gain weight, in spite of the carcinoma. Such patients have been observed.

The diagnosis of carcinoma of the lung can be made by the direct method if a biopsy of the growth can be obtained; otherwise it is made only by the indirect method of ruling out all other diseases. Often exploratory thoracotomy must be resorted to for diagnosis.

A biopsy can be obtained by several methods. Bronchoscopy may reveal a tumor mass from which a biopsy can be made. The possibility of diagnosing the disease by bronchoscopic biopsies has been reported at 78 per cent by Holinger (6) and at 62 per cent by Overholt (11). It should be remembered that a high percentage of the cases with positive bronchoscopic biopsies are inoperable. Clerf (1) believes that only 10 per cent of such cases diagnosed by bronchoscopic biopsy are operable. In the present series positive bronchoscopic biopsies were secured in 14 per cent. Of the patients with positive biopsy obtained by the bronchoscope, pneumonectomy was accomplished in 50 per cent. Clerf has recently shown that the cytologic study of bronchial secretions obtained through the bronchoscope will give a positive diagnosis of carcinoma in 82.4 per cent of the patients. Most important, however, he was able to make a cytologic diagnosis of carcinoma in 21 per cent of patients where there was no bronchoscopic evidence of carcinoma, either by observation or by biopsy. In the present series the result of examinations of bronchial secretions obtained by the bronchoscope have not been nearly as good as those reported by Clerf. The biopsy may be obtained by the punch or aspiration method. For fear of spreading the disease through the chest wall this method is recommended only in cases where concomitant disease makes pneumonectomy impossible. In such patients, it is good to know the exact diagnosis, even though surgical treatment cannot be offered. Where a patient is 70 years old or older the punch or needle biopsy is indicated since one would hesitate to subject such a patient to an exploratory thoracotomy; however, if a positive diagnosis of carcinoma were made, pneumonectomy would surely be indicated. A biopsy of distant metastasis can often be obtained; if this procedure shows carcinoma, it will not only make a positive diagnosis, but it will prevent a useless thoracotomy or pneumonectomy. Virchow's gland in the neck is commonly involved by metastatic carcinoma of the lung and should always be carefully searched for when carcinoma of the lung is suspected or diagnosed. A biopsy showing carcinoma can frequently be obtained by careful pathologic study of the pleural fluid (7), but again a positive biopsy obtained in this manner only saves the patient from a useless operation. It does not meet the challenge of early diagnosis.

The indirect diagnosis of carcinoma of the lung is made by symptomatology, physical findings, roentgenographic examination, and the elimination by direct diagnosis of such diseases as bronchiectasis, pulmonary tuberculosis, pneumonia, and similar conditions. The symptomatology as described will put the examiner on his guard for carcinoma. The physical examination will contribute little. The roentgenographic appearance of carcinoma of the lung is so variable that one can only suspect the presence of the disease by this method. Nevertheless, roentgenography, if used in mass examinations or routinely on all hospital admissions, offers the best method of early diagnosis of carcinoma of the lung. Of course, every patient with a complaint of chest symptoms should have a roentgenographic examination of the lungs. Roentgenograms will be of little help if all abnormal shadows found are not correctly diagnosed. If an accurate diagnosis cannot be made, an exploratory thoracotomy should be performed. There are two roentgenographic findings that often indicate carcinoma of the lung: the presence of a round or rounded opacity, two centimeters or larger in size; and atelectasis of the lung, a lobe, or a part of a lobe that cannot be explained by some acute process or cause. A positive diagnosis of bronchiectasis by bronchogram, the presence of a positive sputum or the diagnosis of pneumonia does not definitely rule out carcinoma of the lung.

In the presence of undiagnosed pulmonary disease or abnormal roentgenographic findings of the chest that cannot be satisfactorily explained, an exploratory thoracotomy should be done. With proper anesthesia, an exploratory thoracotomy is usually safe. In the present series exploratory thoracotomy was done seventeen times, with no death resulting from the operation or its effects. With the chest open, the operator occasionally will not be able to make a positive diagnosis. However, there is little likelihood of mistaking carcinoma of the lung if one is present. Occasionally a biopsy followed by a frozen section, with a prompt histologic diagnosis, is of great value to the operator in making a diagnosis while the chest is open.

Once a diagnosis of carcinoma of the lung has been made or exploration has been decided upon, it becomes imperative that the signs of inoperability be sought out carefully in order to avoid a useless operation. These signs are all evidenced by spread of the growth beyond the confines of the lung, either by local extension or by metastasis. Metastasis may occur anywhere in the patient's body; sometimes it can be found by careful physical examination. A thorough search must always be made for Virchow's gland. If abnormal lymph glands are discovered anywhere or if subcutaneous nodules are found, they should be examined for carcinoma. Metastasis to bone can be ruled out by roentgenographic examination. If a rib is shown to be eroded, the tumor often should be considered inoperable. However, occasionally the ribs and surrounding tissue can be removed in a block dissection together with the lung. If the ribs and vertebrae are eroded, the growth is inoperable. Nerve lesions may occur, and they are always the sign of local extension or metastasis beyond the lung, usually signs of inoperability. Vocal cord paralysis generally occurs on the left side since the recurrent laryngeal nerve courses posterior to the aortic arch on the left and thus has a long course through the thoracic cavity. Paralysis of the phrenic nerve may occur and will manifest itself by a high diaphragm with absence of motion. Horner's syndrome may be present and results from invasion of the first and second dorsal sympathetic ganglia and the sympathetic trunk. The opposite of Horner's syndrome must not be confused with the condition, since it results from stimulation instead of paralysis of the sympathetic trunk and its first two dorsal sympathetic ganglia. The Pancost syndrome may be present and indicates a much more extensive invasion by the neoplasm than Horner's syndrome. It, of course, indicates invasion of the first dorsal and sometimes eighth cervical nerve roots, as well as the sympathetic trunk with the first and second dorsal ganglia. The presence of pleural fluid is usually a sign of inoperability and indicates metastasis to the pleura. The size of the tumor may also be taken as an indication of inoperability. A tumor mass that fills half the chest cavity on one side generally is beyond surgical help. Extreme emaciation often is a sign of metastasis even though such metastatic lesions cannot be demonstrated.

The treatment of carcinoma of the lung is early pneumonectomy. Lobectomy will not permit satisfactory removal of the regional lymph glands. Radiation therapy, either roentgen ray or radium, seems useless, even from the palliative point of view. When the carcinoma of the lung has metastasized or spread beyond the confines of the lung, palliative resection is rarely, if ever, indicated.

Pneumonectomy is a well established and safe treatment for carcinoma of the lung. Today the greatest obstacle to treatment is late diagnosis. The mortality and morbidity of pneumonectomy would be greatly reduced and the postoperative longevity considerably increased if the diagnosis were made earlier in the course of the disease. Many operative deaths occur from the endeavor of the surgeon to accomplish the impossible by removing a growth that has encroached upon the pericardium and surrounding tissues. If the surgeon accomplishes the removal of the extensive growths, the morbidity after operation is increased and the survival period free of recurrence is greatly shortened. Accordingly, early diagnosis will greatly reduce the menace of carcinoma of the lung.

SUMMARY

1. The early diagnosis of carcinoma of the lung is a challenge to the medical profession.
2. The challenge of early diagnosis of carcinoma of the lung can be partially conquered if physicians in all categories will consider the disease from a simple clinical point of view.
    a. Asymptomatic neoplasms of the lung can be detected by roentgenography. If routine roentgenographic examination of the chest is carried out relentlessly there will be fewer patients who have their first symptom as a result of distant metastasis.
    b. Any patient over 35 years of age with a chronic pulmonary complaint or symptom should be suspected of having carcinoma of the lung unless otherwise proved.
    c. Patients with pulmonary symptoms should be accurately diagnosed and in the presence of undiagnosed pulmonary disease or unexplained roentgenographic abnormalities, exploratory thoracotomy should be used freely.

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MULTIPLE PRIMARY NEOPLASMS

A Summary of the Literature and Report of a Case of Four Primary Neoplasms with Complete Autopsy Findings*†

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The occurrence of four primary malignancies in one person is extremely rare, unless one considers the finding of multiple foci of malignant neoplasms in the skin, urinary bladder and gastro-intestinal tract as primary malignancies.

One may exclude these latter groups or consider multiple epidermal carcinomata, multiple papillomata of the bladder and polyposis of the gastro-intestinal tract as essentially one process (Muller, Bargen, etc.) (2, 24). In this case the finding of four primary malignancies, which fall into the criteria stated below, has been reported only ten times. It was first reported by Leyden (20) in 1904 and cited by Gotting (12) in 1909. Here there were six primary neoplastic sites, the largest number of primaries ever reported in the literature exclusive of skin, bladder, and gastro-intestinal tract neoplasms. This was followed by reports in the literature by Goetze (9) (1913), Lauda (19) (1925), Luchsinger (22) (1930), two by Bargen and Rankin (2) (1930), Goldman (11) (1944), Holland (15) (1945), by Mallory, as editor, in the Cabot Clinic (1946), and by Black and Howe (5) (1946).**

Warren and Gates (31) have presented an exhaustive statistical study of the literature and reported 111 cases of three or more malignancies, of which 28 were definitely stated as being of four or more foci. In their study, the skin and gastro-intestinal tract are notorious for the frequency in which multiple neoplasms are found.

Many neoplastic diseases present themselves as multiple lesions. It is fairly common, as noted above, to find multiple skin cancers, multiple papillomas of urinary bladder and multiple neoplasms of the gastro-intestinal tract.

Ewing (8) has stated that if one examines the outside limits of a tumor, cellular changes towards a "precancerous" and preinvasive cystologic picture can be discerned. Thus, "carcinoma in situ" may occur at widely

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** A case was reported by Hayward in 1945 as four primary neoplasms in an 82 year old male, viz., adenocarcinoma of prostate, embryonal carcinoma of left testicle and two separate lesions of the skin . . . the perineum and left groin. The multiple skin lesions, however, eliminate this from the scope of our communication.

separated points in an organ or tissue and "thus produce multiple tumors separated by areas of normal tissue" (Slaughter) (29). However, multiple tumors which occur in different organs and tissues, suggest a tumor diathesis in the individual, with a greater susceptibility to additional anaplastic lesions in an individual already afflicted. This has been suggested and supported by authors on this subject like Bugher (6), Lombard and Warren (21), Warren and Gates (31), Kilgore (17), White (32), etc.

Because of its rarity and particular interest, we present the case history and autopsy report on a man, aged 54, who presented four metachronous malignancies.

CASE REPORT

E. M. H. (5478), aged 53, was first seen on October 22, 1934.

Family history revealed that on the maternal side one female cousin, one male cousin, and an aunt had died of cancer. On the paternal side, two sisters and two brothers, cousins of the patient, also died of cancer.

Past history was essentially negative except for pneumonia twenty years before and an attack of gonorrhea at the age of 18.

The present illness dated back to July 1934, when he suddenly passed bloody urine which stopped just as suddenly after three days. This was painless, but associated with frequency day and night, some burning on micturition, and some urgency. Abdominal examination at this time was negative as to any palpable mass. The external genitalia revealed no evidence of any pathology. Rectally, the sphincter was of good tone. The prostate was of normal size and consistency, as were the seminal vesicles. There were no areas of stony hardness suggestive of malignancy of the prostate. The voided specimen of urine was bloody. Albumin was one plus, sugar negative, specific gravity 1.018, reaction acid. Microscopically, the urine showed many red blood cells and an occasional white blood cell. On cystoscopy, 75 cc. of residual urine was obtained. The bladder was of good tone and had a capacity of 300 cc. There was a slight intravesical bulging of the prostate posteriorly in the form of a small median lobe. Behind the trigone on the right floor of the bladder was a villous mass, springing from a broad pedicle and covered by blood clots. The actual size of this was about 3 cm. The edges around the base were slightly injected. A diagnosis of malignant papilloma of the bladder was made, and fulgurations were prescribed. Thereupon, the tumor was fulgurated on several occasions, completely obliterating the neoplasm. On December 3, 1934, he developed an acute retention and had to be catheterized. A retention catheter was placed in the bladder. The patient was again cystoscoped and a small active tumor was seen on the left side of the floor of the bladder. The tumor which had been on the right side had now disappeared. The tumor was fulgurated and a piece of blood clot was removed with a rongeur after the performance of a meatotomy. A diagnosis of a malignant papilloma of the bladder was made. After an afebrile period with absence of hematuria, the patient was discharged on December 13, 1934. A second admission to the hospital was on March 25, 1935. Since his previous discharge from the hospital, the patient complained of difficulty in urination with nocturia and occasional hematuria. On the day of admission, he had the desire to void but was unable to do so. In this state of acute retention, he was admitted to the hospital. An abdominal examination revealed no palpable tumor mass, but the patient was very obese, so that it was impossible to be certain about any definite palpable mass. A rectal examination revealed a prostate of normal size and consistency. No areas suggestive of malignancy were present. A plain roentgenograph of the abdomen showed a large right kidney, with the psoas muscle on the right not clearly defined. Cystoscopy was performed and many blood clots were evacuated from the bladder. There was no intravesical bulging of the prostate. Blood was coming from the right ureteral orifice. The capacity of the right kidney pelvis was found to be 30 cc. The urine from the left kidney was clear. A ureteropyelogram on the right showed the kidney to be below the crest of the ilium. The pelvis was not dilated, but all the calyces were dilated. The two lower calyces, however, showed definite separation, the lowest one being dilated and slightly compressed, while the terminal portion was compressed and thinned. The impression was of a right renal neoplasm possibly of epithelial type. A roentgenologic examination of the chest showed no evidence of metastasis. In view of the large number of clots in the bladder, a suprapubic cystotomy was performed. Bloody urine was seen coming from the right kidney. There was no intravesical bulging of the prostate. On April 16, 1935, a right extra-fatty capsular nephrectomy was performed. There was a small nodule of tumor tissue seen in the renal vein. An examination of the kidney showed it to be very nodular in the lower half which was covered with a thick layer of fatty capsule. A piece of nodular tumor tissue extended down into the renal vein and was excised. There seemed to be no infiltration of tumor tissue to the fatty capsule. The postoperative course was uneventful. The patient was discharged on the forty-second postoperative day (May 27, 1935) as improved.

The gross pathologic report showed a right kidney measuring 16 x 9 x 7 cm. and lacking its true capsule. It was grayish-brown in color, of firmer consistency in the lower pole than in the upper. The nodules were yellowish and extended into the lower two-thirds of the kidney. There were some areas of depression and some very small cysts. On bisection, the kidney seemed to be the seat, in the lower two-thirds, of numerous small circumscribed yellowish nodules measuring 2 mm. to 1.8 cm. in diameter. These were firm and not connected with the pelvis or calyces but seemed to be in most instances bordering on the mucous membrane of the calyces. This seemed to extend from the pelvis clear out to the cortex. In the lower third of the kidney, numerous similar nodules were seen in the cortical tissue.

Microscopic pathology: Section one was of kidney tissue. There was acute, subacute, and inflammatory exudation in the glomeruli. Throughout the cortical portion of the kidney there was frequent leucocytic infiltration, obscuring the tubular portion of the kidney. There was bloody extravasation throughout the tubular portion of the kidney with marked regenerative changes. Section two was of kidney tissue invaded by growth. The latter showed a heavy supporting fibrous tissue framework, and from this framework there was an off growth of fibrous tissue structure from which a framework supported all the contents. The growth was very cellular, imitating tubules. The cells themselves, lining the so-called tubules, were rather pale staining, fine, granular with numerous clear spaces within them. The nuclei were small and very dark. Frequently, the growth had invaded the fibrous tissue stalk which supported it. The diagnosis of carcinoma of kidney (Grawitz type) was given April 16, 1935.

Following discharge from the hospital on May 27, 1935, the patient slowly regained his strength, vigor, and weight. There was no dysuria, frequency or nocturia, nor any hematuria or acute retention. Twenty days before his last admission (October 29, 1936) to the hospital, he developed pain and tenderness in his lower abdomen. This was followed in two days by chills and fever to 102.4°. The stools were tarry. No fresh blood was seen. There were no urinary complaints during this period of observation. A physical examination on admission revealed a very obese male, presenting a round, cystic, and moderately tender mass in the suprapubic region. There were many naevi scattered over the neck and chest. Over the left shoulder, there was an ulcerative granulomatous lesion 2.5 cm. x 2 cm. in size.

The patient's abdomen became distended, and a Wangensteen was set up without relief. The urea on admission was 184 mgms. per cent, but this soon stabilized to about 90 mgms. per cent. During the last two days he complained of pain in the right upper chest and a diagnosis of pneumonia was made. On the evening of November 4, 1936, the patient went into sudden cardiac failure and developed pulmonary edema. He died soon thereafter.

An autopsy was obtained and reported as follows:

The body was that of an obese, well nourished, well developed elderly male who weighed 185 pounds. Over the left shoulder was an ulcerative, granulomatous lesion which measured 2.5 x 2 cm. This lesion had obviously been present for some time as its margins were quite ragged. On microscopic section of this lesion, one could see the transformation of normal skin with a good deal of chronically infected tissue present in the corium to ulceration of the squamous layer; in the latter region, there was a definite downgrowth of pegs of cells of deeply staining nuclei of the basal cell type. The cells were arranged in whorls and contained a moderate number of mitoses. The infiltration extended deeply into the subcutaneous tissues.

An examination of the thoracic viscera revealed a homogeneous solid metastasis in the right lower lung, which on microscopic section contained numerous closely packed cells with foamy cytoplasm and nuclei which were rather pale and reticular and contained an occasional mitotic figure. The right upper lobe revealed a lobar pneumonia.

The abdominal viscera revealed a liver which contained white hardish nodules, one just above the edge of the right lobe and several scattered throughout the right lobe of the liver. These metastases, on microscopic section, showed a definite glandular arrangement of cells with little cytoplasm, but large reticular nuclei with numerous mitotic figures.

In the region of the large intestine and appendix, there was a large abscess cavity. At a point 20 cm. from the recto-sigmoid, the bowel had a white cartilaginous appearance, and on the upper margin were numerous hard white segregated nodules which appeared to be tumor tissue. The mesenteric lymph glands below the involved area were also completely infiltrated with tumor tissue. Microscopic examination of this tumor mass revealed tumor cells arranged in a glandular fashion. The tumor infiltration extended through the entire thickness of the bowel wall. The picture was consistent with the diagnosis of adenocarcinoma of the sigmoid. The remaining left kidney was enlarged and revealed a cyst at the hilum measuring about 2.5 cm. in diameter.

An examination of the bladder showed it to be normal in size; its wall was not thickened. There was no evidence of prostatic obstruction, ulcers or diverticuli. A close examination of the bladder revealed no recurrence of the papillary carcinoma for which the patient was successfully treated by fulguration in 1934.

Final post-mortem diagnosis:

*1. Adenocarcinoma of the sigmoid with perforation
2. Pelvic abscess
3. General peritonitis (colon bacillus)
4. Metastases of adenocarcinoma in liver
*5. Right nephrectomy (hypernephroma)
6. Metastases of hypernephroma in right lung
7. Cardiac hypertrophy

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* Denotes the four primary neoplasms.

8. Chronic myocarditis
9. Arteriosclerosis
10. Coronary sclerosis
11. Lobar pneumonia (right upper lobe)
12. Cyst of the left kidney
13. Chronic glomerular nephritis
14. Obesity
*15. Basal cell carcinoma of skin over left shoulder
16. Benign prostatic hypertrophy
*17. From the patient's history, a papillary carcinoma of the bladder was successfully treated by fulguration in 1934, and verified by a study of the tissue microscopically.

COMMENT

Billroth (3, 4) first reported multiple malignancies in 1860. In 1869 he formulated three criteria for the establishment of a diagnosis of multiple primary malignancy. These criteria were: (1) each neoplasm must have an independent histologic appearance; (2) each neoplasm must arise in a different situation; and (3) each neoplasm must produce its own metastasis.

These criteria formulated by Billroth (3, 4) have been overtaken by the progress of medicine. More exact diagnostic procedure, the utilization of the roentgen-ray, contrasting media, and the use of electric diagnostic instruments, have enabled the physician to make early diagnoses before the development of visible metastasis, gross or microscopic, and this frequently resulted in complete extirpation of the neoplasm immediately in question. Thus, in addition to early diagnosis, better therapeusis and longer survival following the diagnosis of malignant neoplasm has exposed these individuals once again to the diseases of later life, i.e., anaplasia.

The criteria of Billroth (3, 4) enumerated above for multiple primary malignancies have been found to be too rigid and have now generally been disregarded by workers in this field for a more realistic view to conform to our rapid advances in diagnoses and therapeusis. Reported cures in patients with four primary malignancies have reached the literature several times, having been reported by Bargen (2) twice in 1930, by Holland (15) in 1945, and by Black and Howe (5) in 1946. Nevertheless, only from a post-mortem examination after the death of these cases will it be known if their reported cases can really be reported as such.

For these reasons, the following criteria for diagnosing multiple primary malignancies have been adapted: (1) The presence of neoplastic growths at separate and distinct sites, (2) the cell type, histologically, must show evidence of the aberrant growth of a different tissue or organ, (3) the possibility of one of the lesions being a metastatic growth must be rigorously excluded, (4) the several lesions need not present themselves at the same time, i.e. may be synchronous or metachronous, (5) metastasis need not be present either grossly or microscopically. Thus it is apparent that only the intrinsic tissue characteristics of the neoplasms must differ.

The incidence of multiple carcinomas varied among the different investigators who have reported studies on malignancies and varied from 0.2 per cent by Gotting to 7.8 per cent by Burke. These various studies are tabulated below and show an overall incidence of 2.14 per cent of reported carcinoma autopsies as being multiple.

TABLE I

Incidence of Multiple Malignancy

AUTHOR	YEAR	POST MORTEMS WITH CARCINOMATA	MULTIPLE CARCINOMA	PER CENT
Gotting	1909	1,000	2	.2
Seecof	1924	170	1	.58
Orr	1930	1,046	8	.77
Angevine	1930	910	4	.44
* Warren & Gates (summary)	1932	29,740	546	1.84
Hurt & Broders	1933	2,124	71	3.34
Bugher	1934	983	28	3.1
Schreiner & Wehr	1934	11,212	307	2.7
Burke	1936	583	46	7.8
Kirschbaum & Shively	1938	10,870	25	1.77
Stalker, et al	1939	2,500	113	4.52
Peller	1941	5,876	270	3.9
Hellendall	1943	685	30	4.33
Totals		67,699	1,451	2.14

* In Warren and Gates statistical summary, 713 additional cases of multiple carcinomata were collected as individual reports or otherwise but not included in a postmortem series. This raised their reported cases from 546 as noted above to 1,259.

These statistics are in close agreement with Warren and Gates (31) who reported that in their survey of the literature of 29,740 malignancies, 546 were found to be multiple, an incidence of 1.84 per cent—of these, 28 cases had four or more foci (including multiple foci in skin and gastro-intestinal tract), an incidence of 0.094 per cent. (115,813 post-mortem examinations were found to have 15,988 malignancies, an incidence of 13.8 per cent.) They could not explain the greater incidence of multiple malignancies in America, which they found to be 3.9 per cent. Warren and Gates (31) further state that they consider the true incidence of multiple carcinomata as 2 to 4 per cent of all malignancies, and also that a definite predisposition or susceptibility to cancer is present in certain individuals.

TABLE II

AUTHOR & YEAR	AGE	SEX	LESION AND ORGAN
1. Leyden 1904		F	Carcinomata of uterus, cervix, both breasts, stomach and skin of back.
2. Goetze 1913	75	M	Adenocarcinomata of stomach and prostate, four carcinomata of the descending and sigmoid colon. Also present was polyposis of intestines. Carcinoma simplex of rectum.
3. Lauda 1925	54	M	Epithelial carcinoma of tonsil and of esophagus, carcinoma of bile duct and adenocarcinoma of rectum.
4. Luchsinger 1930	89	M	Carcinomata of forehead and bronchus, hypernephroma of kidney, osteochrondroma of lung.
*5. Bargen & Rankin 1930 (case 4)	59	M	Carcinomata of caecum, transverse colon, splenic flexure and sigmoid.
*6. Bargen & Rankin 1930 (case 6)	46	F	Carcinomata of ascending colon, carcinomata of splenic flexure and sigmoid, sarcoma of uterus.
7. Goldman 1944	60	F	Medullary carcinoma of left breast, post-radiation carcinoma of chest wall with recurrence, transitional cell carcinoma of cervix, papillary adenocarcinoma of rectum.
8. Holland 1945	55	F	Adenocarcinoma of right breast, carcinoma of thoracic esophagus, basal-cell carcinoma of left cheek, annular carcinoma of transverse colon.
9. Mass. General	78	M	Colloid carcinoma of gall bladder, epidermoid carcinoma of bladder, adenocarcinoma of rectum, and adenocarcinoma of prostate.
10. Black and Howe	49	F	Adenocarcinoma of right breast, adenocarcinoma of left breast, adenocarcinoma of uterine fundus, and adenocarcinoma of ascending colon.
11. Goldstein & Rubin	54	M	Papillary carcinoma of bladder, hypernephroma of right kidney, basal-cell carcinoma of skin over left shoulder, adenocarcinoma of sigmoid.

* This case was diagnosed by a pathologic examination of the operative specimens. No post-mortem examination was reported to exclude the possibility of the existence of polyposis in the remainder of the large intestine. Bargen and Rankin, however, considered multiple polyposis, but considered these cases as being primary lesions of the colon.

White (32) explains this predisposition to multiple carcinomata in certain individuals on a basis of the interaction of a functionally mature gene bearing unit for cancer inheritance with one for localization together with the necessary environmental stimuli.

On the other hand, Peller (26), on the basis of his statistical analysis, states that "a cured tumor leaves protection against other cancers." This, on the whole, does not appear to substantiate the facts. Lombard and Warren (21) have also shown that the incidence of multiple carcinomata in those with cancer of the skin was 5.9 per cent or 2.2 per cent higher than the series Warren reported with Gates (3.7 per cent).

The incidence of multiple neoplasms in one individual seems to be increasing. Four cases of four primary malignancies have appeared in the literature since 1944. This may be the result of a more general microscopic examination in the post-mortem room; to more frequent "cures"; to a longer life expectancy; to better public health educations; and/or to the greater diagnostic acumen of the physician.

The four primary malignancies reported so far are summarized in table II.

SUMMARY

1. A review of the literature of multiple primary neoplasms is presented.
2. Multiple malignancies are being encountered more frequently, but four primary lesions are still very rare.
3. Eleven cases of three or more primary neoplasms have been found in the literature including the case reported by the authors.
4. A case of four primary malignancies is reported. The primary sites are as follows:
   a. Malignant papilloma of the bladder
   b. Adenocarcinoma of the sigmoid
   c. Right hypernephroma
   d. Basal-cell carcinoma of the skin
5. A complete autopsy report is included.
6. Criteria for making the diagnosis of multiple primary malignancies are given.
7. Multiple lesions of the large intestine and skin, such as multiple malignant papillomata of the bladder, are not included as multiple primary malignancies of that organ.
8. A table summarizing the existing cases of four or more primary malignancies in one individual is included.
9. Half of the total number of such cases reported have reached the literature since 1944.

10. Multiple malignancies are occurring and are being diagnosed more frequently because of better diagnostic aids, more efficient therapeusis, and longer survival following the diagnosis of malignancy with frequently reported "cures". More general post-mortem examinations, longer life, and more generalized public education on health also help to reveal multiple malignancies.

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CANCER OF THE BREAST*†

The Present Status of Irradiation

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The purpose of this paper is to review briefly the prognosis of cancer of the breast, with special emphasis on the present status of irradiation. The study is based on personal experience and a very exhaustive review of present day literature.

FACTORS IN PROGNOSIS

Several factors are important in estimating the prognosis of cancer of the breast, but the most important are as follows: 1. Microscopic grading, popularized by Broders, has been of limited value because no two pathologists will grade the same slide alike, and a slide from a different area of the same tumor may have a different grade; thus, even though a high grade or rapidly growing tumor will probably metastasize earlier, it is not always possible to predict this from microscopic grading. 2. The pathologic classification of breast cancer is of some value in estimating the prognosis; for example, a medullary carcinoma may be fairly rapid in growth, a scirrhous cancer usually grows more slowly, and colloid cancers, even though few in number, grow slower than any other type; thus the type of tumor is also of some value in estimating the prognosis. 3. The extent of the local growth, i.e., size of the growth, edema of the skin of the breast, fixation of the tumor to the skin, fixation of the tumor to underlying chest wall, secondary cancer nodules in the skin over the tumor, ulcerations of the skin, and finally the presence of metastases, are much more important as prognostic signs than either microscopic grading or the pathologic classification of the tumor.

CLASSIFICATION OF BREAST CANCER

The local extent of the growth is so important in the prognosis of breast cancer that all authors, both radiologists and surgeons, should classify their cases in the future into clinical stages. Probably the most popular classification at present is that of Portman (10), which is as follows:

Stage I. Tumor—localized in breast and freely movable.
Skin—not involved.
Metastases—none.

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Stage II. Tumor—localized and movable.
Skin—not involved.
Metastases—few in axilla.

Stage III. Skin—edematous; brawny red induration not caused by infection; extensive ulceration; or multiple secondary skin nodules.
Tumor—diffusely infiltrating the breast; fixation to the chest wall; edema of the breast; or secondary tumors in breast.
Metastases—many axillary nodes; or fixed axillary nodes.

Stage IV. Skin—as in any other stage.
Tumor—as in any other stage.
Metastases—many axillary or supraclavicular nodes; or any distant metastases.

RESULTS OF SURGERY

Before a detailed discussion on the importance of irradiation therapy for cancer of the breast, it is necessary to summarize briefly the five year survivals by surgery alone. This has been a little difficult to do as almost every clinic now uses irradiation; however, after reviewing numerous articles from both large and small clinics, it should suffice to give the results of two of the largest clinics, which are as follows: Harrington (6) 70.2 per cent cures without axillary nodes, and 24.3 per cent cures with axillary nodes; and Adair (1) 74.2 per cent cures without axillary nodes, and 47.3 per cent with axillary nodes. The Memorial Hospital series, as here reported by Adair (1), represent a highly select group of cases, and it is not likely that the average clinic could report such a favorable figure (47.3 per cent cures) for cancer of the breast with axillary nodes treated by surgery alone.

IRRADIATION THERAPY

Irradiation therapy for cancer of the breast was first given in America January 29, 1896 by E. H. Grubbe (3) of Chicago. Since that time it has gone through various stages of popularity, but up until about 1930 the therapy was insufficient, equipment was poor, and most radiologists knew very little about therapy. Any statistics before that time, whether good or bad, have very little value at present. There have been so many improvements in the technic of roentgen therapy, i.e., increasing the total dose, elimination of posterior portals, decreasing the kilovoltage to decrease lung fibrosis, and recognizing the value of the daily dose method of therapy, that statistics on irradiation from papers written only in recent years will be used in this paper.

Indications for Irradiation: Irradiation is used as a curative agent in a small group of early (stage I) cases. Preoperative or postoperative roentgen therapy should supplement surgery in all stage II and III cases. Irradiation, either alone or combined with testosterone or irradiation sterilization, should be the accepted treatment for all stage IV cases, metastases, and local recurrences.

Stage I Cancer of the Breast: Numerous clinics have reported and all agree that surgical removal will cure 70–85 per cent of early cancers of the breast. The improvement in five year survivals by the addition of either preoperative or postoperative roentgen therapy has been very little. Harrington (6) reports an improvement of 5.2 per cent, and Adair (1) reports a slightly lower figure, 2.6 per cent. However, it is probably true that some of these early cases had unrecognized positive nodes in the axilla at operation, and properly should have been classified as stage II, as it is doubtful if roentgen therapy adds very much for early localized cancer with no axillary nodes present.

There are a few early operable cancers of the breast that, for one reason or another, i.e., age, poor condition, cardiac disease, or even refusal to permit operation, have to be treated entirely by irradiation. In most clinics roentgen therapy alone, probably the easiest method, is used and usually with only moderate success. Most clinics report that an average of 40 per cent of these early cases can be carried beyond the five year limit. When one considers that the average duration of life for breast cancer without any treatment has been estimated at 40.5 months by Daland (2), it is obvious that roentgen therapy alone has a low curability rate; but, as a palliative agent, it does prolong life for months, or even years, and keeps the patient comfortable longer by checking the progress of local growth, thus putting off the time for metastases.

The combination of interstitial radium (either radon seeds or radium element needles) and roentgen therapy has a curability rate almost equal to radical removal for early cancer of the breast. The results from this procedure are about the same as simple mastectomy, probably a little better, but not quite equal to the results of radical removal with axillary dissection. Keynes (7), London, and Meland (9), California, report 71.4 and 67 per cent, respectively, five year cures by the combination of interstitial radium element needles and roentgen therapy. Keynes (7) has probably had more experience than most American radiologists, thus American statistics on this procedure are of little value. The author, however, has used this procedure in a few select cases and finds that if the cancer is early, small in size, well localized, and with no axillary nodes present, it is a valuable procedure for poor risk (too poor for simple mastectomy) cases, and it can be done easily with local anesthesia in an office or out-patient clinic.

Simple mastectomy for early, but poor surgical risk, cancer of the breast has an average five year survival rate of about 60 per cent. In this country it is much more popular, and most Americans believe it gives better results, than the combination of interstitial radium and roentgen therapy popularized by the British. It is especially valuable for large and bulky growths, where the amount of irradiation necessary to control such growths might result in radiation necrosis. From the literature, it was obvious that every few years many surgeons would become discouraged with the results of radical mastectomy and, because of the low operative mortality and morbidity rate, they would again try to popularize simple mastectomy. It should be noted that the curability rate of simple mastectomy is about two times better than roentgen therapy alone, but far below radical mastectomy and axillary dissection combined with roentgen therapy.

Stage II Cancer of the Breast: Cancer of the breast with an occasional positive lymph node in the axilla (Stage II) has an average curability rate of 40–50 per cent by radical removal and routine postoperative roentgen therapy. The results of three of the largest clinics are as follows: Harrington (6) 29.4 per cent, Adair (1) 41.8 per cent, and Marshall and Hare (8) 37 per cent. However, this is not quite accurate as they included all operable cases with positive axillary nodes (Stages II and III), and thus their figures are lower than the general average.

Roentgen therapy as a supplement to surgical removal is now fairly well proven, and there are few clinics that do not recommend it. A fairly extensive review of the literature shows the average curability rate of Stage II cancer of the breast has been increased 10–15 per cent by the addition of routine roentgen therapy. It is true that Harrington (6) noted only a 5 per cent improvement, but this low figure is not the concensus of opinion throughout this country or Britain. Local recurrences in the breast area and axilla have decreased by the routine use of roentgen therapy as a supplement to radical removal. Roentgen therapy also causes a delay in local recurrences after operation; thus, even though the patient later dies of cancer, life has been prolonged and made more comfortable. Theoretically, irradiation destroys many of the cancer cells, and the fibrosis produced locks up some of the residual cancer cells, thus delaying local recurrence and metastasis.

Simple mastectomy combined with intensive roentgen therapy for the axillary nodes is occasionally necessary for bad risk cases. However, the curability rate is not equal to radical removal because of the known fact that roentgen therapy alone is not equal to dissection for the control of axillary metastases.

Stage III Cancer of the Breast: Cancer of the breast with fairly extensive local involvement, i.e., edema of the skin of the breast, skin nodules in breast area, secondary nodules in the breast, fixation of the tumor to the skin, fixation of the tumor to the underlying chest wall, or local ulceration, accompanied with many axillary nodes (Stage III) is the borderline operable group, and the average five year survival rate is about 20–30 per cent. To evaluate the prognosis in Stage III cancer of the breast by surgical removal alone, or removal combined with roentgen therapy, it is necessary to remember that Daland (2), Boston, estimated the average duration of life of untreated breast cancer was 40.5 months, 22 per cent lived past the five year limit with no therapy, and 5 per cent lived ten years with no therapy. Irradiation therapy as a supplement to radical removal has probably increased the average curability rate of this group of cases about 10 per cent.

There are still two schools of thought in regard to operation for Stage III cancer of the breast: 1. Harrington (6), the chief exponent of the radical view, recommends operation and postoperative roentgen therapy for all these cases, and his five year survival rate for all Stage II and III (and probably a few Stage IV) cases combined is 29.4 per cent. 2. Conservative surgeons, notably Haagensen and Stout (4), believe operation is not only not indicated in many Stage III cases but may even shorten life, and they cite 109 cases of the borderline operable group to prove the point. Many surgeons would do well to study carefully their criteria of operability for Stage III cancer of the breast. Many radiologists will classify these borderline cases as inoperable or Stage IV and recommend palliative roentgen therapy. This means the possibility of cure is nil. The odds are in favor of operation and irradiation in most borderline cases, even though the five year salvage is small.

Irradiation not only adds about 10 per cent cures to Stage III cancer of the breast, but it is extremely valuable as a palliative agent. It will decrease ulceration, secondary infections will improve, pain is decreased, and in many cases life is prolonged, economically more useful, and in general more comfortable.

Stage IV Cancer of the Breast: Fortunately inoperable cancer of the breast is on the decrease. Every clinic has noted it. The educational campaign of the American Cancer Society has been largely responsible for the improvement, even though a gradual increase in operable cases was noted years before their work began.

Authors do not agree exactly on just what criteria indicate inoperability in cancer of the breast, but in general the indications are as follows: cancer of the breast with supraclavicular nodes; all cases of breast cancer with distant metastases; cancer of the breast associated with pregnancy; all cases with edema of the arm or edema of over one-third of the breast area; all local recurrences after operation; and many Stage III cases that have two or more signs of local extension or fixation.

Radical surgery is contraindicated in all Stage IV cancer of the breast, thus roentgen therapy as a palliative agent is the procedure of choice in the majority of such cases. Occasionally local excision combined with roentgen therapy is a better palliative procedure than roentgen therapy alone for foul-smelling ulcerations and many bulky growths that would require too much irradiation to reduce their size. The purpose of irradiation in these cases is to reduce the size of the growth, decrease the ulceration, clear up infection, decrease the pain, and thus hope to prolong life and economic usefullness. There are no cures by roentgen therapy and there are no reliable statistics of just how long life is prolonged in such cases. It is known that about 40 per cent of the Stage I cases can be carried beyond five years by roentgen therapy alone, 20–25 per cent of the operable cases (Stage I, II, and III) can also be carried beyond five years by roentgen therapy alone, but Daland (5) has estimated that 22 per cent of all cases (Stages I to IV) will live past five years with no therapy; thus it is difficult to evaluate statistics in advanced malignancy but irradiation has a delaying effect on cancer and must prolong life.

Local recurrence after operation is a common indication for palliative irradiation in Stage IV cancer of the breast. Local recurrences depend upon several factors but the most important are as follows: (1) the extent of the local growth at the time of operation, i.e., ulceration of the skin, secondary skin nodules, or fixation of the tumor to skin or underlying chest wall; (2) the number of axillary nodes present at operation; (3) the thoroughness of the radical removal and axillary dissection, even the amount of time taken for the operation; and (4) the thoroughness of the postoperative roentgen therapy. The average recurrence rate reported is about 20–30 per cent. The total dosage of roentgen therapy given is a very important factor in recurrences; for example, Haagensen and Stout (5) used 800 r over the breast, axilla, and supraclavicular areas and their recurrence rate was 22.8 per cent, while Marshall and Hare (8) used 2400 r to the same areas and their recurrence rate was only 8 per cent. Increasing the total dosage of roentgen therapy in recent years is probably one of the most important factors in the definite improvement in five year cures of cancer of the breast.

Roentgen therapy is indicated for all local recurrences, and usually is a good palliative agent. It has a delaying effect on the growth, thus a few cases will live past five years, even though they later die of metastases. Local recurrences often have a tendency to become ulcerated and painful. A course of roentgen therapy may give relief from pain for months. The author has seen palliative relief of several cases of local recurrences at Mercy Hospital, Baltimore, in the past few years.

Roentgen therapy is widely used as a palliative agent for distant metastases in various locations, most commonly for bone metastases. Many of these cases get partial relief of their pain from a light course (1500–2000 r) of roentgen therapy. Pathologic fractures occasionally show recalcification. Some of the most grateful patients seen by radiologists are the group that get relief of pain from vertebral metastases.

PREOPERATIVE AND POSTOPERATIVE ROENTGEN THERAPY

It is now established that roentgen therapy adds 10–15 per cent to the five year survival rate for operable cancer of the breast, but the undecided question is whether it should be preoperative or postoperative. Most surgeons prefer postoperative therapy. This has been more noticeable since Adair's (1) paper in 1943. Surgeons reason that the delay of operation caused by three to four weeks of roentgen therapy, the possible delay in healing of skin reactions, the possibility of metastases from the delay, and the increased difficulty of operation as a result of radiation fibrosis, are strong points against preoperative irradiation. Most radiologists prefer preoperative therapy. They believe preoperative irradiation will destroy a large portion of the growth, do considerable damage to the remaining cancer cells, and thus decrease the local implants of cancer cells at the time of operation. If the cancer cells, which are left in the operative area from the trauma of operation, are practically destroyed from preoperative roentgen therapy they are not as likely to become implanted and start growth again as cancer cells in cases without preoperative roentgen therapy. The question cannot be decided from present day literature, but the results are about as follows: 1. Soiland (12) and Meland (9) estimated that preoperative roentgen therapy increased their five year cures by 17 per cent, making their cure rate for all operable cases (with and without axillary nodes) 47 per cent. 2. Portman (11) reports an improvement of 9.1 per cent in five year survivals in all operable cases by adding postoperative roentgen therapy, making his total cures 47 per cent. 3. Harrington (6) reports 5.2 per cent increase in cures for Stage I and 5.1 per cent increase in cures for all other operable cases by routine postoperative roentgen therapy, making his five year cures total 47.6 per cent. 4. Adair (1) uses routine postoperative roentgen therapy with a 51 per cent five year survival of all operable (Stage I, II, and III) cancer of the breast. Marshall and Hare (8) routinely use postoperative roentgen therapy and have increased their five year survivals to 52.1 per cent, which is 13.5 per cent better than before they started roentgen therapy at the Lahey clinic.

TIME RELATION OF OPERATION AND IRRADIATION

The consensus of opinion is that three to five weeks after the completion of preoperative roentgen therapy is the optimum time for radical removal of the breast. This is about the time of the maximum amount of destruction of cancer cells as it is known they can begin to grow again about the sixth week after irradiation, the skin reaction has usually subsided by this time, and radiation fibrosis usually appears much later and thus it should not interfere with the operative procedure at this time. Occasionally some one recommends immediate operation (within one or two days) after intensive roentgen therapy, but there is no statistical evidence to substantiate such a procedure.

Those men in favor of postoperative roentgen therapy recommend that it should begin within a few days after operation. They hope to destroy any residual cancer cells left in the operative field from the trauma of operation before they become implanted and start to grow again. Immediate postoperative roentgen therapy does not interfere with wound healing, and thus it should be given as early as possible after operation.

SUMMARY AND CONCLUSIONS

A brief summary of the present literature on the prognosis of cancer of the breast, with special emphasis on the present status of irradiation therapy has been presented.

1. The results of surgery and postoperative irradiation are about as follows:

Stage I.—75–85 per cent 5 year cures.
Stage II.—40–50 per cent 5 year cures.
Stage III.—20–30 per cent 5 year cures.
Stage IV.— 0–10 per cent 5 year cures.

2. Roentgen therapy has added very little (2–5 per cent) to the five year survivals of Stage I cancer of the breast. The combination of interstitial radium and roentgen therapy is about equal to simple mastectomy (60–70 per cent cures) for early operable but poor risk cases of Stage I cancer of the breast. Roentgen therapy alone is a good palliative agent but the curability rate is low.

3. Roentgen therapy as a supplement to surgical removal has increased the five year survivals 10–15 per cent for Stage II and III cancer of the breast.

4. Roentgen therapy is the most valuable palliative agent for all Stage IV cancer of the breast, all local recurrences, and all distant metastases, most often bone metastases. It delays the progress of many advanced cases, decreases the percentage of local recurrences, improves ulcerations, and diminishes the pain of metastases.

5. The merits of preoperative versus postoperative therapy were discussed, and from a purely statistical viewpoint, the results were about equal, namely, 10–15 per cent increase in cures by either preoperative or postoperative roentgen therapy; however, there is a growing tendency in most clinics to favor postoperative roentgen therapy.

6. The optimum time for radical removal of the breast after preoperative roentgen therapy is three to five weeks. The optimum time to begin postoperative roentgen therapy is a few days after operation.

7. The most favorable fact noted by the author in an exhaustive review of present day literature on cancer of the breast was that all clinics, both from

this country and others, report that women are seeking advice earlier, and early treatment is probably a better explanation for the definite improvements in results noted than any improvements in surgical technic or irradiation.

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PARAPLEGIA AS THE RESULT OF METASTASIS FROM CARCINOMA OF THE PROSTATE GLAND*†

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Not all patients with carcinoma of the prostate gland show demonstrable metastasis to bones, and not all patients with metastasis have symptoms of pain or obstruction to urination. However, this is the exception rather than the rule.

According to McCrea (6) in a series of 812 patients with carcinoma of the prostate, proven metastasis occurred 345 times, or 42.14 per cent. The most frequent site of the metastasis was the lumbar spine. Sharpe (8) has found that tumor cells which had metastasized to the spine from carcinoma of the prostate appeared to stimulate the endosteal as well as the subperiosteal osteoblasts to activity. This differed from the metastasis to the spine as a result of breast carcinoma. He also found that between the different grades of carcinoma there was little difference in the tendency of the growth to metastasize to bone.

Clarke and Viets (1) report a case in which there was carcinoma of the prostate with metastasis and a complete block of cerebro-spinal fluid. The protein of the spinal fluid was 789 mg. After the block was relieved by the use of stilbestral, the spinal fluid protein dropped to 52 mg. They call attention to the fact that metastatic lesions in the lumbo-sacral spine cause pain in the legs from direct pressure on nerve roots, that the relief of these symptoms may be of more urgent concern than the urinary dysfunction and that many of these patients complain of "neuritis" or "rheumatism" rather than urinary retention.

Huggins and his associates (5) reported a case with paraplegia treated by orchiectomy.

Nesbit and Cummings (7) reported 2 cases of transverse myelitis with complete relief of pain and return of function following orchiectomy.

Out of a total of 122 patients admitted to the University Hospital with carcinoma of the prostate gland, 3 of them had paraplegia. The improvement in these patients was so marked after therapy that a detailed account of their symptoms, findings, and treatment follows:

H. K., aged 70, was admitted July 14, 1942 with inability to stand or walk, general weakness, pains "all over my body," and hematuria. Symptoms had begun in September,

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† Read at the Urosurgical Club, Sleepy Hollow Country Club, Scarsdale, N. Y., October 4, 1947.

1941, with pain and weakness in the lower extremities. Before admission, the patient had been bedridden for some weeks and was using opiates to control the pain. Except for the periodic hematuria, there were few urinary symptoms.

The physical examination showed a pale, emaciated patient, with excruciating pain in the legs and shoulders, with an enlarged liver, and a typically hard, fixed and painful prostate. There was flaccid paralysis of the left leg, and bilateral sensory and motor impairment in both legs. There was marked secondary anemia. A roentgen examination showed evidence of metastasis to the pelvis and spine. There was a small metastatic lesion in the left lower lobe of the lung and pleural effusion. The urine was blood tinged on admission but cleared almost entirely before operation. The acid phosphatase was 110 and the urea nitrogen 50.

A bilateral orchiectomy was done on July 19, 1942. Within twenty-four hours after the operation, the patient reported that the pains had practically disappeared. There was general marked improvement. Two days after the operation the acid phosphatase fell to 3.7. The urea nitrogen went to normal a few days later. Roentgenography three months after castration showed the lung metastasis gone and the pleural effusion absorbed, but no change in the bony metastasis was noted. Within three weeks, the patient could stand without aid; and now, after five years, he is walking without aid and is able to do farm work. He has been on stilbestral, 1 mg., daily, almost constantly since the operation, his only complaint being painless enlargement of the breasts.

E. R., aged 59, was admitted October 25, 1945, with the complaint of urinary urgency, increasing for the past two years. The physical examination was essentially negative, except for a blood pressure of 170 systolic over 100 diastolic, and a large, hard, fixed prostate gland. Roentgenography showed no evidence of bone metastasis.. The acid phosphatase was 0.8. A transurethral resection was done October 31, 1945, the tissue showing adenocarcinoma. The patient's recovery was uneventful, and he was put on ethinyl estradiol 0.05 mg., once a day. He was admitted again on September 4, 1946, complaining of general weakness and dysuria. An examination showed the prostate to be smaller and softer. There was no roentgenologic evidence of metastasis to bony structures. The acid phosphatase was 0.45, the blood pressure 142 systolic over 76 diastolic. The neurologic examination was negative. On September 6, 1946, a bilateral orchiectomy was done. Within twenty-four hours there was marked improvement in the symptoms. The patient's recovery was uneventful. He returned home, and continued to take stilbestral, 1 mg. daily.

He was admitted for the third time on June 6, 1947, almost moribund with abdominal distention, incontinence of bowel and bladder, weakness and numbness of both legs, which he had had for three weeks prior to admission. The prostate was the same as before. Roentgenography now showed marked evidence of metastasis to the pelvis and spine. The acid phosphatase had remained normal at 0.61. There was practically no sensory sensation from the xiphoid down, and no motor sense. The leg reflexes were absent. After abdominal decompression by means of suction and enemas, the patient was given stilbestral 5 mg. three times a day by mouth, starting the fifth day after admission, or as soon as syphonage was stopped. By June 15, or the ninth day after admission, the patient was able to control his bowels and bladder, and sensation had returned in the legs. By July 1, he was up in a wheel chair, and two weeks later was able to walk. When last seen, September 1, 1947, or two months after the last admission, the patient was doing well, walking a little, gaining weight and strength under stilbestral 5 mg. three times a day without any breast changes or other untoward symptoms.

A. N., aged 76, was admitted January 11, 1941, with an acute retention of urine. Aside from the urinary tract, the general examination was negative, and a diagnosis of benign prostatic hypertrophy, Grade II, was made. A suprapubic prostatectomy was done, with an uneventful recovery. Sections of the removed prostate showed a benign, prostatic hypertrophy. The patient was admitted a second time on July 20, 1947, with an acute retention of urine. There was marked weakness in the legs, with impairment of the sensory and motor impulses, also secondary anemia and infected urine. A rectal examination indicated a small, hard, fixed mass in the prostatic bed. The acid phosphatase was 1.0. Roentgenography of the bones, which had previously been clear, now showed evidences of metastasis to the bony pelvis, spine and ribs. The patient was treated by indwelling catheter for the retention. He was not castrated, because of objections, but was given stilbestral in increasing quantities up to 15 mg. a day. After six weeks, he could void freely and had improved sufficiently to walk out of the hospital. He is still improving in general strength.

These cases afford a striking example of the improvement to be expected following what Huggins calls antiandrogenic therapy.

COMMENT

In view of the finding by Sharpe (8) that in bone metastasis there is endosteal osteoblastic activity, it is assumed that the condition produces a pressure myelitis. It is, therefore, probably as a result of this pressure that paraplegia at times is produced from pressure directly on the spinal cord. Many of the leg pains so commonly associated with cancer of the prostate are evidently caused by pressure on the nerve roots supplying these extremities.

The treatment of advanced carcinoma of the prostate gland by steroid substances with estrogenic properties or by removal of testicular tissues, has been under observation for more than five years. It is believed that in addition to the testes, the adrenals and the pituitary exert a hormonal influence in the body. It would seem, therefore, that the first attack in these advanced cases should be to control, as far as possible, the general hormonal influence, reducing the androgenic stimulus by giving estrogenic substances first, and if this is not effective, resorting to castration. British urologists seem to have this opinion. On the other hand, many American urologists believe that castration decreases the amount or activity of the androgens and is indicated before estrogenic therapy is used in order to make it more effective. Still others believe that if the androgenic stimulus of the testes is removed, this function is gradually taken up by the adrenals or the pituitary.

Huggins (4) states that two factors are of significance in determining the effectiveness of orchiectomy.

1. The tumor must be androgen-dependent.
2. The testes must contribute functionally significant amounts of the total amount of androgen.

He feels that castration is superior to the neutralization of the androgens by the administration of estrogens. Results as a rule are excellent in giving at least temporary relief to these patients, although about one-fifth of the patients are not benefited by either castration or estrogenic therapy. Certain failures must be caused by inadequate dosage of the estrogens. The most impressive results are to be seen in the advanced cases.

In the hands of the authors serum acid phosphatase tests have not been reliable, either in establishing the diagnosis of metastasis or in showing the extent of metastasis. When the test is positive, it is confirmatory of clinical diagnosis of carcinoma of the prostate with metastasis. It shows a high reading in advanced cases and falls promptly after castration or the institution of estrogenic therapy.

SUMMARY

1. Three patients out of 122 with carcinoma of the prostate gland developed paraplegia as a result of spinal metastasis.
2. The paraplegia probably resulted from pressure on the cord from endosteal bony outgrowths.
3. All three cases were relieved by castration or the use of estrogenic substances or both.
4. The first patient reported has had more than five years of relief of symptoms.

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REPORT OF A CASE OF SYRINGOMYELIA COMPLICATING PREGNANCY*†

J. TYLER BAKER, M.D. AND JULIUS STOLL JR., M.D.

BALTIMORE, MARYLAND

Organic disease of the spinal cord in the parturient woman is relatively rare. In a review of the English literature, only one case of syringomyelia complicating pregnancy could be found.

Syringomyelia is a disease of the spinal cord, and/or brain stem, which was first outlined as a clinical syndrome by Kahler and Schultze in 1887. The pathologic aspect is that of extensive central gliosis and often of cavity formation throughout the neuraxis, with extension in a variable direction to the periphery. Such destruction produces signs of local damage at the site of the lesion, and also an evidence of abnormality below the level as a result of long tract involvement.

REPORT OF A CASE

The patient was a thirty year old, colored female, gravida 3, para 2, who was admitted to the obstetric division of the Baltimore City Hospitals on November 10, 1946 in early labor. Her expected date of confinement was November 13, 1946. Her past medical history revealed that twelve years previous to admission she had noted that she was clumsy with her hands, and that they began to contract. This was more marked on the right and had progressed slowly to a marked flexion deformity. She also stated that she frequently burned the fingers of both hands, because she could not feel heat or pain. She denied spontaneous pain, bladder symptoms, or any complaints referable to her lower extremities. She had previously two full term pregnancies, one five years, the other two years before this admission. The duration of labor in her first pregnancy could not be determined; but it terminated spontaneously. Her second pregnancy terminated spontaneously after a total labor of seven hours. Her prenatal course had been uneventful.

A physical examination revealed a thirty year old, colored female at term. There was a marked claw-like deformity of both hands. The fingers presented several ulcerations on the dorsal surfaces; and the spine showed a very evident upper thoracic kyphosis, but there was no pes cavus. On looking to the right, there was a marked gross nystagmus, and a less marked nystagmus on looking to the left. Over the right side of the face there was a decrease in pain and cold perception. The cranial nerves were otherwise normal. On the right side was some weakness and wasting of the spinati, serrati, and deltoid muscles, a moderate weakness and some wasting of the biceps, brachial radialis, and extensors of the wrist, as well as marked weakness and wasting of the triceps and flexors of the wrist. Bilaterally, there was a marked weakness and wasting of the small muscles of the hands, and a slight diffuse weakness of the right lower extremity, with some increase in tone. The right foot was everted. There was decreased perception of pinprick and cold on the entire right side of the body with the exception of the right arm, which presented a

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* From the Baltimore City Hospitals.

† Received for publication October 15, 1947.

complete loss of perception of pinprick and cotton wool. However, at the fifth sacral level, all modalities of sensation were intact (sacral sparing).

REFLEXES

	Right	*Left*
Biceps	Normal	Normal
Radial	Absent	Absent
Triceps	Absent	Absent
Patella	Exaggerated	Exaggerated
Achilles	Exaggerated	Exaggerated
Abdominals—Upper	Absent	Absent
Lower.	Absent	Absent
Plantar.	Flexor	Flexor

The anterior-posterior roentgenograph of the cervical and upper thoracic spine revealed some right convex scoliosis in the thoracic region, which was otherwise normal. Myelography done postpartum revealed no defects after injection of 6 cc. of pantopaque. The cerebrospinal fluid examination showed: a clear fluid, normal dynamics, an initial pressure of 130 mm. of water, no cells, normal Pandy, negative Kolmer, 26.6 mgm. per cent of protein, and a normal colloidal mastic test. The urinalysis was normal, the hematocrit was 37 and the blood serologic test for syphilis was negative.

After a total labor of two hours and eight minutes, during which she received sodium pentobarbital gr. 1½ and gas-oxygen analgesia with her pains, she delivered a full term, living male child weighing 7 pounds 6½ ounces from a left occipito-anterior position spontaneously. Her postpartum course was uneventful and there was no evidence of bladder atony or bowel incontinence.

DISCUSSION

A patient pregnant and at term presented evidence of involvement of the anterior horns of the cervical enlargement, as revealed by muscle wasting, a dissociated type of sensory loss on the right side of the body with the exception of the right upper extremity which presented complete anesthesia, a nystagmus, early signs of a spastic paraplegia, a slow progressive course, and no signs of an extra medullary lesion on myelography. There seems little doubt that this was clearly a case of syringomyelia, involving the cervial enlargement of the spinal cord. She had been delivered spontaneously of three full term living children, with no obstetric complications. This case substantiates the observation of other authors: that cord disease of this nature and severity per se is not an indication for the interruption of pregnancy or abdominal delivery.

CONCLUSIONS

1. Syringomyelia is a rare complication of pregnancy.
2. The mechanism of labor per se was in no way influenced by syringomyelia in this patient.

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CHRONIC ULCERATIVE COLITIS*†

WALTER LINCOLN PALMER, M.D.

INTRODUCTION BY DR. MAURICE FELDMAN

It seemed appropriate that Dr. George B. Eusterman, professor of Medicine, Mayo Foundation, University of Minnesota, should have been selected to give the first Friedenwald Lecture. Now we call upon Dr. Walter Lincoln Palmer to give the second.

Dr. Walter Lincoln Palmer is the past president of the American Gastroenterological Association, having served during the 1946–47 term. Dr. Palmer has done considerable investigative work and has also made many original contributions in gastroenterology. He is professor of Medicine at the University of Chicago and Chief of the gastroenterologic service at Billings Hospital. To many in the audience in the gastroenterologic specialty, Dr. Palmer needs no formal introduction, and to many others he is known by his work. It gives me great pleasure to present Dr. Walter Lincoln Palmer, who will speak on "Ulcerative Colitis."

It is a privilege to give the second annual Julius Friedenwald lecture. In my first contacts with Dr. Friedenwald, his ability as an inspiring teacher and a gifted clinician was readily apparent. As the years passed and I learned to know him better, his friendly smile and kindly word of encouragement made each meeting with him a memorable one for me. It is therefore a great pleasure to join with you tonight in honor of Dr. Friedenwald. I thank you most sincerely for the opportunity.

It was altogether fitting and proper that the first lecture should have been given, as it was a year ago, by Dr. Friedenwald's old friend, Dr. George B. Eusterman, who in June of this year also received the annual Julius Friedenwald award of the American Gastroenterological Association. These two men had much in common: a wide knowledge of clinical medicine and especially of gastroenterology, excellent clinical judgement, an ability to keep abreast with newer developments and to contribute to scientific advance, a capacity for warm and lasting friendships.

The onset of ulcerative colitis may be acute or insidious. In the acute form, the diarrhea usually ceases after a few weeks or months, to be followed by subsequent exacerbations. This tendency to spontaneous remissions and exacerbations is one of the characteristic features of the disease. The rectum and rectosigmoid are involved in the initial stages. The feces at this time is usually formed. Localization of the process and mildness may persist for years. The disease may advance retrogradely up the bowel and thus may precipitate at any time an attack of acute diarrhea.

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* From the Department of Gastroenterology, Billings Hospital, Chicago, Ill.

† Abstract of the "Julius Friedenwald Memorial Lecture," presented at the University of Maryland, School of Medicine, on October 23, 1947.

Proctoscopic evidence of the disease is found in 97 per cent of cases; roentgenologically the rectum and sigmoid are observed to be affected in 58 per cent, the entire colon in 30 per cent.

The relationship between the extent of the disease and the severity of symptoms is not a direct one, although as a rule the greater the involvement, the more severe the symptoms. Profuse bleeding and tenesmus may be seen with a localized lesion; and on the contrary, an extensively diseased colon may be discovered in an apparently well person, passing formed stools. The relationship between the duration of the disease and the severity of the process is also not direct. The demonstration of the characteristic proctoscopic changes in the rectal mucosa is essential for the diagnosis. The diffusely friable edematous mucosa is almost pathognomonic.

Perforation of the bowel with resultant peritonitis is the most serious complication and the chief cause of death. Perirectal fistulae and absesses may be most troublesome. Inflammatory polyposis is very common and in about 2 per cent of cases leads to malignancy.

The two most promising theories of the etiology are the infectious and the psychogenic. Infection and inflammation play an important role in the disease process. Difference of opinion arises as to the identity of the organism and the mechanism of the infection. The differing results obtained in ulcerative colitis with the various chemotherapeutic agents also suggest that the process may be maintained, if not initiated by a variety of organisms.

With the psychogenic theory, the problem is also a quantitative one as to what extent and under what circumstances do the emotions play a role in the disease. There seems to be some significant correlation between the emotional life of a patient and the symptoms. Of interest is the question whether emotional stress can initiate ulcerative colitis or precipitate a relapse. The difficulties in the interpretation of the psychogenic manifestations should be emphasized.

The principles of the treatment of ulcerative colitis, such as bed rest, and a diet low in residue, high in calories and in protein is standard. Parenteral amino acid and glucose feeding may be of great value in the acute phase. Antispasmodics and sedatives are helpful. The sulfonamides given in adequate amounts do change the fecal flora; but does this influence the course of the disease is a question. It is difficult to give a convincing answer because the disease spontaneously remits, which beclouds all attempts at evaluation. In many instances the administration of the sulfa drugs does help in checking the character and number of stools.

The evaluation of penicillin has been equally difficult. It may be useful in certain cases. The combination of sulfa and penicillin drugs may aid in checking the disease.

The role of psychotherapy in the treatment of ulcerative colitis is as difficult to appraise as the value of antibiotic drugs. Psychotherapy cannot be prescribed in daily doses, nor can the results be weighed and measured. From this point of view the disease itself may be looked upon as evidence of an unresolved conflict and of the patient's complete inability to solve the problem successfully. Supportive reassuring psychotherapy may yield extraordinary results in some instances.

The chief indication of surgery is intractibility. Ileostomy may restore these individuals to a life of normal activity. However, as we learn more about the disease, the number of patients requiring ileostomy seems to be decreasing. Colectomy is indicated for polypoid hyperplasia, stricture formation, and similar conditions. Surgery is not a treatment of the disease itself, but of certain complications.

It is difficult to state the results of therapy in statistical terms. One hundred and forty one patients were studied and followed for variable periods of time. There have been 19 known deaths, 13 without benefit of surgery and 6 in spite of surgery. Twenty-five patients must be listed as unimproved. Two-thirds of our patients have been able to get along well and to carry on satisfactory lives. Only 5 patients were classified as cured by which is meant absence of symptoms for five years or more, with normal bowels as seen both proctoscopically and roentgenologically. Few diseases tax the patience and ingenuity of the physician more than does ulcerative colitis; few demand more of the combined science and art of medicine; and yet few yield better returns in terms of grateful patients.

OBSTETRIC CASE REPORT*†

E. D., a 40 year old, para 12–0–0–12, was admitted to the hospital, at term but not in labor, after a normal prenatal course in one of the county clinics, because of a persistent transverse lie of the fetus. Previous past, personal, and obstetric histories were normal. The presentation was confirmed by roentgenographic examination (the fetal skeleton appeared normal). Placentography showed the placenta to be implanted near the fundus of the uterus. Except for the abnormal presentation, the general physical and obstetric examinations were within normal limits. Laboratory studies were normal. Repeated gentle external cephalic versions were successful, but in each instance the fetus spontaneously reverted to the transverse lie, in spite of the application of various abdominal binders. It was decided to keep the patient in the hospital for further observation and a decision as to her subsequent management.

DISCUSSION

Here, the obstetrician is dealing with a grand multipara, at term, with a persistent transverse lie. The greatly relaxed abdominal wall undoubtedly contributed to the etiology of this abnormal presentation. Two other possible etiologic factors, namely, placenta previa and fetal abnormality, had tentatively been ruled out by the roentgenographic examination. There were two methods of delivery to be considered. First, in view of the persistent transverse presentation, abdominal delivery was discussed. Second, since the patient was in the hospital under careful observation, external cephalic version could be performed immediately upon the onset of labor, with the fetal head held over the pelvic inlet until it became well engaged in the pelvis.

MANAGEMENT

On the twelfth hospital day, without previous symptoms, the patient began to bleed bright red blood in small amounts without pain. There were no uterine contractions; there was no tenderness, and the fetal heart was normal. After preparations had been made for any eventuality, a sterile pelvic examination under the "double set-up" was performed in order to rule out placenta previa, in spite of the placentographic report. The cervix was 75 per cent obliterated, deeply scarred by old multiple lacerations and was about 3 centimeters dilated, but no pathologic lesions were noted. Placental tissue could not be palpated, and the membranes were found to be intact. Since the patient was at term and the source of bleeding had not yet been determined, it was deemed advisable to induce labor.

After an external cephalic version had brought the head over the pelvic inlet, the membranes were ruptured, allowing the amniotic fluid to escape

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* From the Department of Obstetrics, School of Medicine, University of Maryland.

† Received for publication November 7, 1947.

slowly until most of it had drained away. A scalp clamp was then applied to the fetal scalp. There was a three hour latent period before the onset of labor, during which time the patient was carefully observed. The fetus was found to remain in a longitudinal lie. Because of the multiparity of the patient, pituitrin was not given to stimulate labor. From the onset of labor, progress was rapid, the patient being delivered spontaneously after one hour and fifty minutes, without apparent laceration, of a full term living child, weighing 8 lbs. 12 oz. The third stage was uneventful; the placenta was normal. No source of bleeding was discovered. The total duration of labor was two hours. The fetal scalp clamp, which had been removed when the head had descended to the mid pelvic plane, had caused no damage to the fetal scalp. The subsequent course of both mother and baby was normal.

This case illustrates a practical use for the fetal scalp clamp.

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* The names listed above are officers for the term beginning July 1, 1947 and ending June 30, 1948.

NEWS ITEMS

The following Massachusetts alumni have recently applied for membership in the Massachusetts Medical Society:

John D. Barnes, New Bedford; U. of Md. 1943.
Edward F. Briggs, Truro; B.M.C., 1906.
Richard L. Fowler, Spencer; U. of Md., 1943.
Leonard T. Kurland, Brighton, Boston; U. of Md., 1945.
Rocco L. Sapareto, Haverhill; U. of Md., 1943.

Dr. James F. Sasscer (U. of Md., 1934) of Upper Marlboro, Maryland, has been elected Chief of Staff of the Prince George's General Hospital.

Dr. Bricey M. Rhodes (U. of Md., 1922) of Tallahassee, Florida, was elected Third Vice President of the Florida Medical Association at the Annual Meeting of the Association, April 23, 1947.

Eli Lilly & Company have awarded a grant of $9000 to Dr. John C. Krantz, Jr., Professor of Pharmacology, School of Medicine, University of Maryland, for the purpose of pursuing studies on hypertension. Dr. Joseph G. Bird, a graduate in medicine from this institution, has been appointed Fellow to prosecute these studies.

The Ohio Chemical & Mfg. Co. has awarded a fellowship of $3500 to the Department of Pharmacology of the School of Medicine, University of Maryland, for the purpose of continuing the study of volatile anesthetic agents. Doctor Krantz, Professor of Pharmacology, is in direct supervision of these studies.

The Board of Trustees of the United States Pharmacopoeial Convention has renewed the appointment of Dr. Frederick K. Bell as Research Assistant in Pharmacopoeial Problems in the Department of Pharmacology of the School of Medicine, University of Maryland. The problems of immediate study are the standardization of digitalis and heparin sodium.

Dr. H. A. B. Dunning, Chairman of the Board of Hynson, Westcott and Dunning, has given $1000 to the Department of Pharmacology, School of Medicine, University of Maryland, for general research purposes under the direction of the head of the department.

Dr. H. A. B. Dunning, Chairman of the Board of Hynson, Westcott and Dunning, has presented an oil painting to the Department of Pharmacology, School of Medicine, University of Maryland, depicting the discovery of digitalis by William Withering. Ceremonies of the unveiling of the painting took place on October 6. Dr. C. Jelleff Carr, Associate Professor, was Master of Ceremonies.

OBITUARIES

Carpenter, Charles Augustus, Caldwell, N. J.; B.M.C., class of 1909; aged 68; died, July 6, 1947, of carcinoma of the prostate.

Coughlin, Alfred G., Athens, Pa.; B.M.C., class of 1905; aged 63; died, May 1, 1947, of hypertensive cardiovascular disease.

Craven, William Wilhelm, Charlotte, N. C.; class of 1903; aged 68; died, May 15, 1947, of coronary thrombosis.

Davis, Robert Galen, Baltimore, Md.; P & S, class of 1882; aged 88; died, July 19, 1947, of a hip fracture received in a fall.

Ewing, Edward Hilts, Stoughton, Mass.; P & S, class of 1897; aged 74; served during World War I; died, March 8, 1947.

Franklin, Joseph Powell, Cumberland, Md.; class of 1921; aged 50; died, June 15, 1947, of carcinoma of the liver.

Goldmann, Bernhard Alexander, Pittsburgh, Pa.; class of 1922; aged 51; died, April 7, 1947, of coronary disease.

Goodall, Edwin Baker, Boston, Mass.; class of 1909; aged 65; died, August 6, 1947.

Hanna, Benjamin Sargent, Norfolk, Va.; P & S, class of 1912; aged 72; served during World War I; died, July 14, 1947, of arteriosclerosis and gastric ulcer.

Harris, Charles Edward, Hyannis, Mass.; B.M.C., class of 1897; aged 78; served during World War I; died, May 21, 1947.

Higgins, Gregory L., Carbondale, Pa.; P & S, class of 1910; aged 63; died, June 25, 1947, of fat embolism and a fractured femur received in a fall.

Holland, Stanley Hall, McKeesport, Pa.; P & S, class of 1914; aged 59; died, July 22, 1947, of coronary occlusion.

Johnson, Charles Herbert, Camden, N. J.; class of 1909; aged 65; died, July 12, 1947, of injuries received in a fall.

Kisner, Allan Oscar, Bethlehem, Pa.; P & S, class of 1896; aged 82; died, July 5, 1947, of arteriosclerosis and bronchopneumonia.

Krager, John Joseph, Baltimore, Md.; class of 1922; aged 52; died, recently of coronary thrombosis.

McGarrell, John J., Baltimore, Md.; class of 1908; aged 64; died, May 12, 1947, of cerebral hemorrhage and hypertension.

Milbourne, Leander Burgess, Baltimore, Md.; class of 1898; aged 67; served during World Wars I and II; surgeon aboard the S.S. Santa Isabel on which he died while at sea near Panama on April 16, 1947.

Miller, Charles Albert, Las Cruces, N. Mex.; P & S, class of 1897; aged 76; died, May 13, 1947, of chronic myelogenous leukemia.

Nash, Charles Patterson, Alderson, W. Va.; P & S, class of 1884; aged 88; died, July 12, 1947.

Norwood, Vernon Lee, Baltimore, Md.; class of 1885, aged 85; died, July 13, 1947, of cerebral arteriosclerosis and right hemiplegia.

Plecker, Walter Ashby, Richmond, Va.; class of 1885; aged 86; died, August 2, 1947, of injuries received when he was struck by an automobile while crossing the street.

Podlewski, Stephen S., Steubenville O.; B.M.C., class of 1895; aged 74; died, April 8, 1947, of heart disease.

Roberts, Charles Wesley, Atlanta, Ga.; class of 1906; aged 63; died, July 28, 1947.

Roberts, Joseph John, Baltimore, Md.; class of 1916; aged 58; died, July 16, 1947, of coronary occlusion.

Rowland, Edward Gould, Trenton, N. J.; B.M.C., class of 1903; aged 68; died, April 9, 1947, of lymphosarcoma.

Schlappich, Charles Edwin, Birdsboro, Pa.; B.M.C., class of 1908; aged 65; died, May 14, 1947, of injuries received in an automobile accident.

Shatswell, James A, Beverly, Mass.; P & S, class of 1890; aged 80; died August 26, 1947.

Sutliff, Stephen Dana, Shippensburg, Pa.; P & S, class of 1901; aged 69; died, July 16, 1947, of injuries received in an automobile accident.

Thompson, William Robert, Fort Worth, Texas; P & S, class of 1888; aged 84; died, May 1, 1947, as the result of a fall.

Warden, Frank Ryan, Adamsville, R. I.; P & S, class of 1889; aged 81; died, May 28, 1947, of coronary thrombosis.

Wood, William Francis, Bedford, Va.; P & S, class of 1893; aged 84; died, April 19, 1947, of myocarditis.

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COMBINED THEOGLYCINATE AND PENICILLIN AEROSOL IN THE TREATMENT OF SEVERE ASTHMA*†

Preliminary Report

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Numerous reports have appeared in the recent literature on the use of antibiotics by inhalation methods in the treatment of pulmonary infections (1). In 1946, Prigal (4) stated that aerosolization of aminophylline solutions for the relief of dyspnea in bronchial asthma had been successful. The relief of the dyspnea associated with bronchial asthma encountered in the use of this preparation (5), and the favorable response of the infection of intrinsic bronchial asthma to penicillin aerosol (2) suggested that a combination of the two might be effectual in the control and treatment of patients in status asthmaticus. In March, 1947, this method of treatment was begun as part of a larger study on the toxicity, clinical response, and comparative efficacy of theoglycinate** with other preparations containing theophylline. Detailed reports of this work will be published at a future date. However, because of the remarkable success encountered in the combined theoglycinate and penicillin aerosol therapy, and because of the complete absence of any serious toxic effects, a preliminary report was considered advisable.

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† Read before a staff meeting of the University Hospital, Baltimore, Maryland, December 18, 1947.

** Theoglycinate is theophylline and sodium glycinate, 50 per cent of which is active theophylline. Supplied by the Brayten Pharmaceutical Company, Chattanooga, Tennessee.

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Of the three methyl derivatives of xanthine that are in clinical use, namely, caffeine, theobromine, and theophylline, the last exerts the greatest influence on the cardiovascular system and the bronchial tubes. Unfortunately, theophylline is only slightly soluble, and requires the formation of a double salt in order to render it available for assimilation. This double salt has a definite tendency to cause gastric irritation, thus limiting its field of usefulness. It occurred to Krantz and his associates (3) that further solubility studies on theophylline might be in order to obtain a less irritating end-product. In the course of their investigations, they found that it was readily soluble in a sodium glycinate solution. The glycinate has the advantage over the acetate and salicylate in that it contains an amino group which prevents precipitation of theophylline in the stomach by neutralization of the gastric acidity. This has made possible the administration of large doses of theophylline orally in the form of sodium theophylline glycinate over a period of time without production of symptoms of gastric irritation.

The treatment of asthma separates itself naturally into two divisions, the treatment of symptoms and the treatment of cause. There is actually a third division; for status asthmaticus presents a problem of such severity and such seriousness that heroic measures, usually unnecessary, become imperative.

In this article there is little, if any, concern with the usual methods directed toward the treatment of cause. The entire emphasis is placed upon symptomatic therapy in the face of a severe asthmatic attack. Also, severe asthma is being dealt with, frequently of the grade known as status asthmaticus; and this, as has been mentioned before, presents problems of its own. Ordinarily, asthma is thought to be caused by specific agents, by infection, or by a combination of the two. Therefore, the treatment of cause in the infective cases would be directed toward the eradication of the causative infection.

However, in status asthmaticus, or in the more severe cases of asthma that do not reach this level of severity, infection of an acute nature has usually occurred. Consequently, symptomatic treatment then demands a combination of etiologic as well as symptomatic measures. One of the first things that is necessary is the widening of the bronchial lumina, so that a free passage of air may be obtained with the associated proper egress of mucus. However, ignoring the acute infection present would doom the foregoing measures, even when temporarily successful. The relief obtained would be of a transitory type, which is all that is obtainable with the average symptomatic drug. In order to insure prolongation of such relief, prompt measures must be taken to eliminate the acute infection. Fortunately, the newer antibiotic agents have been dramatically successful in eliminating acute infections in a gratifyingly large percentage of patients presenting these serious symptoms.

Therefore, to recapitulate, the successful treatment of the severe asthmatic episode, whether it has reached the level of status asthmaticus or not, is faced with two demands: first, the widening of the bronchial lumina for the passage of air and the discharge of mucus, and second, prompt measures to combat the acute infection present. The more leisurely handling of other factors must, of necessity, follow the foregoing. Consequently, the first problem is comfort from the standpoint of dyspnea, with as great a degree of return to normalcy as possible, and the second, the carrying out of the many measures available for the long-time control of asthmatic symptoms.

The difficulties encountered in attempts to relieve the prolonged dyspnea in refractory cases of bronchial asthma are well known. In the authors' hands, combined theoglycinate and penicillin aerosol therapy has proved to be more efficacious in the treatment of status asthmaticus than any other combination of drugs now in general use, both from the standpoint of more successful, and of more prolonged relief of symptoms following failure with conventional therapeutic methods. It is presented not as a cure for asthma but as a highly successful method for the handling of severe cases, including status asthmaticus. Cases other than severe bronchial asthma were also treated successfully with this method, but they will receive no discussion here since asthma is the condition under consideration. The authors plan to give this method of treatment further study in these conditions.

Equipment: In almost all cases, the aerosol solutions were administered in a small, transparent canopy* with oxygen or an oxygen-helium mixture for nebulizing the solutions. Three commercial brands of nebulizers** were used, and all were equally satisfactory. The amount of oxygen required to nebulize the solutions varied with the brand of nebulizer used, from 2½ to 5 liters of oxygen per minute. The canopy can be used in various positions, as is illustrated in Figures 1, 2, and 3, depending on the degree of orthopnea present. For mildly dyspneic patients, the apparatus can be placed at the head of the bed (see Figure 1), which then can be elevated as high as 30°, with the patient supine or on his side. With markedly orthopneic patients, the canopy can be placed on the usual hospital bed table and the head of the bed elevated sufficiently to support the patient's back (see Figure 2) Where these tables are not available, the canopy can be placed on the side of the bed and the patient can sit on a chair facing the bed (see Figure 3).

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* Manufactured by The Oxygen Equipment Manufacturing Corporation, New York, New York.

** Breatheasy Distributors, Inc., Seattle, Washington; The Vaponefrine Company, Upper Darby, Pennsylvania; The DeVilbiss Company, Toledo, Ohio.

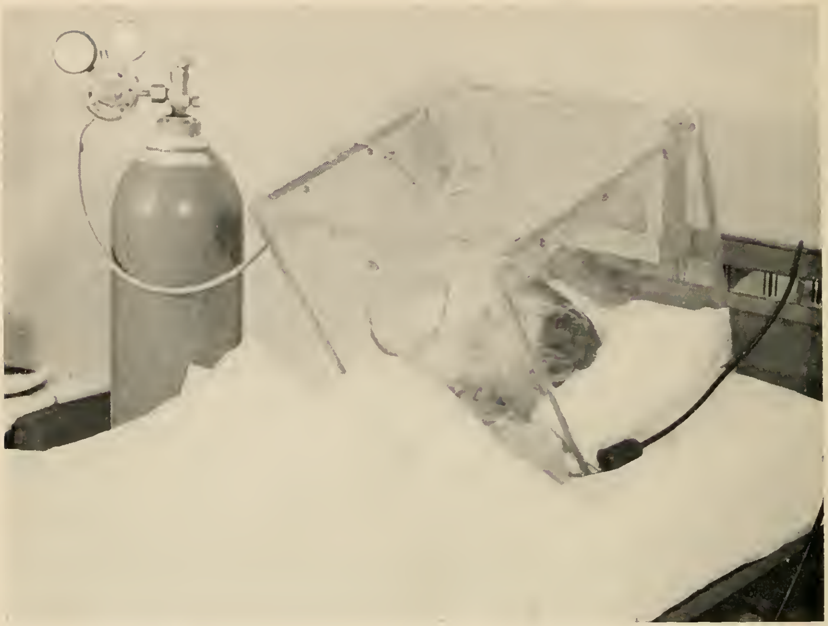

Fig. 1. Demonstration of method of aerosolization with the canopy for the mildly dyspneic patient

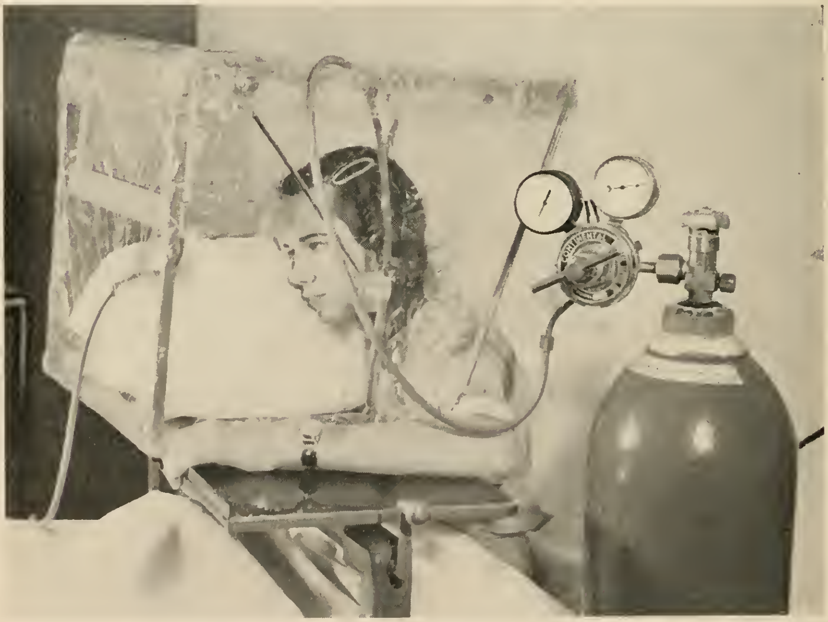

Fig. 2. Demonstration of method for aerosolization with the canopy for the markedly orthopneic patient

Other methods of administration of aerosol solutions have included open hand nebulizers, open nebulizers attached to oxygen tanks, open nebulizers with Y-tubes attached to oxygen tanks, open nebulizers with a mouthpiece, a rebreathing bag, and a Y-tube attached to an oxygen tank, and nebulizers with a regular oxygen mask and tank. Good results have been obtained with all of these methods, but the canopy is by far the most satisfactory, because of the more uniform clinical response, and because of the complete comfort of the patient without the necessity for his handling the apparatus.

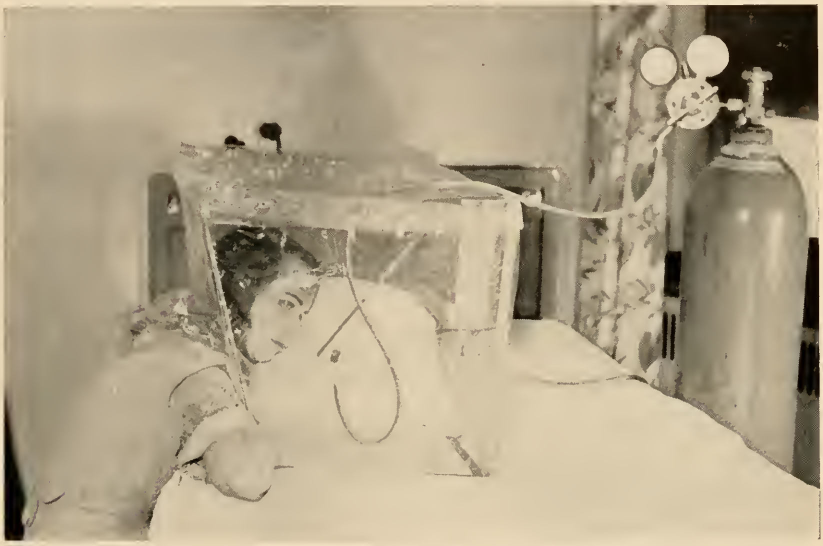

FIG. 3. Demonstration of alternative method of aerosolization with the canopy for the markedly orthopneic patient

There are a number of other definite advantages to supplying aerosol solutions in a canopy. The severely orthopneic patient can be treated in the upright position without adding further to his discomfort. Moreover, the actuely ill or irrational patient is not required to use his limited physical energy or mental concentration to carry out the treatment. It is carried out for and not by him, when he may well be incapable physically and mentally of administering therapy to himself. Also, although the patients were warned that the theoglycinate might momentarily aggravate their dyspnea, none exhibited the claustrophobia common in dyspneic patients administered oxygen in the usual oxygen tent. The complete transparency of the canopy is probably responsible for the elimination of the sense of suffocation so frequently encountered when confining devices are used. In addition, the patient's condition and needs can be readily ascertained at all times without disturbance, and the chest is free and unencumbered so that the frequent examinations that are necessary do not disturb him.

The canopy has been used as successfully with children as it has been with adults. None of the 5 children treated by this method, the youngest of whom was 1½ years of age, ever had to be restrained. It is easy to set up, and simple to run, and most of the patients ran it themselves when sufficient improvement had occurred in their condition. The greatest drawback, clogging of the nebulizers with crystals, was eliminated when the nebulizers were rinsed immediately after each treatment.

Method: In the majority of cases, a 5 per cent solution of theoglycinate aerosol was used, usually 2 cc. every four hours preceding the penicillin aèrosol. In cases with severe dyspnea, continuous aerosol of 5 per cent theoglycinate has been used for twelve and one-half hours and 10 per cent theoglycinate for six hours with considerable benefit and no untoward symptoms. The theoglycinate was followed by penicillin aerosol, usually 20,000 units in 2 cc. of sterile distilled water or normal saline, although as much as 50,000 units has been administered every three hours. The usual time required for aerosolization of 2 cc. of the theoglycinate and 2 cc. of the penicillin was forty to forty-five minutes. Aerosol solutions of both streptomycin, 50,000 units in 2 cc. of sterile saline, and sodium sulfadiazine, 4 cc. of a 5 per cent solution, have been utilized at four hour intervals in conjunction with and in place of penicillin with a satisfactory therapeutic response and no untoward symptoms. Inasmuch as this study was part of a larger clinical study on the effects of theoglycinate, oral theoglycinate was used in most cases. With a gradual increase in dosage, a number of adults have been taking 60 grains daily with a good therapeutic response and no untoward symptoms. In the pediatric age group, daily doses as high as 30 grains have been tolerated.

Additional symptomatic therapy has included butanefrine,* 1 to 1½ cc. (2 to 3 mgm.) intramuscularly every twenty minutes when necessary for dyspnea; penicillin intramuscularly in saline and beeswax; sulfadiazine orally; theoglycinate, butanefrine, and aminophylline intravenously; and adrenalin in oil. Sedation has been accomplished with demerol and sodium luminal administered parenterally, and ether in oil rectally. Therapeutic general anesthetics with ether following cyclopropane induction have also been used.

At the time of this report, a total of 48 cases had been treated with theoglycinate and penicillin aerosol. Of these, 33 had bronchial asthma, and 18 of these were in status asthmaticus. Laboratory data on 10 of the cases of asthma are presented in Table I, and 3 of these are discussed in detail. These cases were selected at random as being representative of the entire group. The results in the rest of the cases have been equally promising, and will be published at a later date.

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* Supplied by the Winthrop Chemical Company, New York, New York.

CASE REPORTS

Case No. 1: This 65 year old white male severe asthmatic was admitted in acute respiratory distress, being cyanotic and orthopneic, on February 20, 1947, having been transferred from another hospital where his asthma had remained out of control for the preceding two weeks despite rigorous therapy. He gave a history of having had constant asthma for the past three months, with little or no relief from the usual therapy. On admission, a physical examination showed typical signs of severe asthma. Transitory relief of the dyspnea was obtained with 1.5 mgm. of butanefrine intravenously, but the respiratory distress returned within ten minutes and the patient was refractory to further treatment. Within twenty-four hours he had become progressively exhausted and appeared critically ill. At this time he was anesthesized with ether for forty-five minutes following induction with cyclopropane. About four hours following the anesthetic he appeared much worse, but thereafter he began to improve gradually clinically and two days later was comfortable at bed rest, although signs of asthma persisted on auscultation. At this time his temperature, which had been normal, rose to 102° F. and he was started on penicillin intramuscularly. His temperature subsided, but because of persistence of signs of asthma on auscultation, he was started on theoglycinate and penicillin aerosol on March 1, 1947. After the first treatment, he was able to lie flat in bed for the first time. At the end of four days of treatment, his chest was clear on auscultation. It remained clear for two days after cessation of aerosol therapy, when signs of asthma recurred on auscultation. An attempt was made, because of the shortage of this equipment at that time, to clear up his chest signs with an open hand nebulizer. When this was unsuccessful after three days, an open nebulizer with an oxygen tank was tried for five days. This likewise was unsuccessful, so the patient was again placed in the canopy for treatment. Within twenty-four hours, his chest was clear on auscultation. It remained so for another twenty-four hours of treatment, at which time he was discharged with directions to take 8 grains of theoglycinate four times daily and 1 cc. of 10 per cent theoglycinate aerosol three times daily, which is self-administered with an open hand nebulizer. Since discharge on March 19, 1947, he has had nine minimal asthmatic attacks in seven months, all readily controlled with 8 to 10 grain doses of oral theoglycinate in addition to his daily dose, which has now been reduced to 20 grains.

Discussion: Attention is called in this case to the severe and prolonged nature of the patient's symptoms, the failure of all other methods to control his symptoms, and the failure of anesthesia to control them completely. The fact that the canopy was effectual when the open method of administration of these drugs met with failure is also noted.

Case No. 2: This 34 year old negro male moderate asthmatic was admitted March 21, 1947, in moderate respiratory distress with the history of a chill, chest pain, and fever ten days before, and continuous asthma for seven days. Fluoroscopy showed a marked patchy infiltration throughout the right chest, except at the extreme costophrenic angle and apex. There was hilar infiltration on the left. This extended upward toward the apex and laterally and downward to surround a solitary lung cyst about 6½ to 8 cm. in diameter and about two-thirds full of fluid. After the first treatment with theoglycinate and penicillin aerosol, the dyspnea was much less marked. After eight hours, the patient was completely comfortable clinically. No parenteral therapy was ever necessary to control his asthma until the oral theoglycinate was stopped on the thirty-fourth day. Within twenty-four hours he developed his first asthmatic attack since admission. This was readily controlled with 2 minims of adrenalin in saline, 1:1,000, and the oral theoglycinate was resumed with no recurrence of asthmatic attacks. The pneumonia was controlled entirely with aerosol penicillin, 30,000 units every four hours for eight days. A second course was begun eighteen days after the first had been completed, in conjunction with postural drainage, with the hope that the process of resolution would be speeded up. No measurable increase was observed. A chest roentgen ray, taken after four aerosol treatments, showed that the fluid level in the lung cyst, which later at operation was shown to have a communicating bronchus, had decreased to half the area of the cyst. Four days later the cyst was empty of fluid. A roentgenograph taken six days after the aerosol treatment was stopped showed a return of the fluid level in the cyst to one-fourth its area. The fluid level continued to rise despite the postural drainage and penicillin aerosol without theoglycinate. The patient was discharged on May 8, 1947, on 10 grains of oral theoglycinate three times daily. He had no asthma until he gave out of theoglycinate. When his supply was renewed, he had no more until after bronchoscopy. He was readmitted on July 17, 1947, for an exploratory thoracotomy. His chest roentgenograph at this time showed increased bronchovascular markings on the right and the cyst three-quarters full of fluid. After six days of preoperative treatment with theoglycinate and penicillin aerosol, the cyst contained no fluid on fluoroscopy. There was none at operation three days later, when a lower left lobectomy was performed. A culture of the cyst at operation was sterile. Three days postoperatively, theoglycinate and penicillin aerosol were resumed because of mild asthma not readily controlled with adrenalin in oil. The patient had one attack of asthma twelve hours after the aerosol treatment was begun, and three days after it was stopped. Both attacks were readily controlled with 2 doses of butanefrine intramuscularly, 2 mgm. each. He developed a thrombophlebitis on the fourteenth postoperative day. At this time his prothrombin time was sixteen seconds with a control of fourteen seconds. This responded readily to specific chemotherapy and dicoumarol. He was discharged August 21, 1947, on 10 grains of oral theoglycinate four times daily. He has had two asthmatic attacks in the three months since, both associated with upper respiratory infections and controlled satisfactorily with extra 10 grain doses of oral theoglycinate.

Discussion: Attention is called to the rapidity with which the pneumonia was controlled by aerosol penicillin; the emptying of the lung cyst following the use of the method under consideration; and the fact that the lung cyst had emptied twice following the use of combined therapy whereas aerosol penicillin alone with postural drainage had been ineffectual. Further, the necessity for the removal of major foci of infection if long-term relief is to be obtained is emphasized by the therapeutic success following the removal of the cyst.

Case No. 3: This 3 year old white female was admitted May 18, 1947, with a typical asthmatic episode associated with vomiting. She was placed in the canopy and theoglycinate followed by penicillin aerosol was started. After one attempt to get out of the canopy, she drifted off to sleep, and her symptoms began to clear almost immediately. Later treatments were accepted without protest and without the need of restraint, and within seventy-two hours she was entirely clear of symptoms. A slight flare-up of the infection and the asthmatic symptoms occurred and was readily controlled.

Discussion: The point of interest here is the ready acceptance on the part of a young child of this method of therapy. This is in marked contrast to the reaction of such young children to the use of tents and other measures heretofore available.

In Table I, laboratory data are presented on 10 cases of asthma, the hospital courses of three of which have been presented and discussed in detail. Seven were severe asthmatics, 3 were moderate. The ages of these 10 cases ranged from 3 to 65 years, with an average age of 43 years. Six white and 4 negro patients are presented, 6 of whom were males and 4 females. The total number of days of hospitalization was two hundred and fifty. The durations varied from eight to eighty-four days with an average stay of twenty-five days. In general, the length of hospitalization varied directly with the length of time evidence of infection had been present and the necessity for surgical procedures. The only untoward symptom, nausea in *Case No. 1* caused both by the too rapid increase in the dose of oral theoglycinate and by the presence of asthma, was promptly relieved when the dose of theoglycinate was reduced and the asthma subsided.

No toxic changes have become apparent in the blood or urine. No decrease from normal has been noted in the prothrombin time of any patient receiving this therapy in comparison with a known normal control, using the Quick method for the evaluation of prothrombin (6). Sputum cultures have been obtained in the majority of cases on admission, and repeated in five to seven days or before initiation of any new chemotherapy. All cases which had *Escherichia coli* in their sputum on admission had previously received penicillin. All cases in which cultures were taken after treatment had this organism in their sputum except one, *Case No. 3.*, on whom a second culture was not taken until thirteen days after the penicillin therapy had been stopped. At this time the culture had returned to the normal flora of the nose and throat. These results correspond in general with those obtained in the remainder of the cases thus treated. Other routine and diagnostic procedures, such as chest roentgenography, venous pressures, circulation times, etc., have not been reported unless abnormal or necessary to the proper evaluation of the case.

The evaluation of the degree of dyspnea present at the time of admission and the degree of therapeutic response was largely based on the presence or absence of cyanosis, the degree of orthopnea, and the chest findings on auscultation. In the presence of cyanosis, the patient was considered severe. He was also considered severe if the accessory muscles of respiration were in use and he was unable to breathe except when sitting bolt upright or hunched forward. If there was any diminution in the intensity of the breath sounds, indicating marked bronchial obstruction, he was likewise considered severe. Cases in which the degree of dyspnea was

TABLE I

Report of Clinical Data on Ten Patients Hospitalized with Bronchial Asthma Receiving Combined Theoglycinate and Penicillin Aerosol Therapy

For comment on each patient see the end of this table

CASE NO.	DIAGNOSIS	DEGREE OF SEVERITY	AGE IN YEARS	COLOR	SEX	DAYS OF HOSPITALIZATION	SERIES OF DRUG	FORM OF DRUG*	HOSPITAL DAY BEGUN	HOSPITAL DAY ENDED	DURATION OF THERAPY IN DAYS	DAILY DOSE*	TOTAL SERIES DOSE*	TOTAL DRUG DOSE*	UNTOWARD SYMPTOMS	LABORATORY											
																Urine				Hematology			Prothrombin Time in Secends			Bacteriology of Sputum	
																Hospital Day	Albumin	WBC	RBC	Hospital Day	% Hb	WBC	Hospital Day	Control	Patient	Hospital Day	Organisms
1	Bronchial asthma	Severe	65	White	M.	27	1	OT	1	3	2	24	44		Nausea	1	0	0	0	1	135	8,000	Not done			4	*Viridans streptococcus*
							2	OT	3	5	2	32	60		Nausea	26	0	0	0	26	95	7,250					*Staphylococcus albus* (moderate number)
	Pulmonary emphysema						3	IMP	4	13	9	240	2,130	2,130	None												
							4	OT	5	6	1	64	56		Nausea												*Staphylococcus aureus* (a few)
							5	OT	6	12	6	24	100		None												
							6	AT	10	15	5	7½	39		None												
							7	AP	10	15	5	200	1,040		None												
							8	OT	12	15	3	32	114		None												
							9	OT	15	28	13	32	408	782	None												
							10	AT	18	18	⅖	7½	3		None												
							11	AP	18	18	⅖	100	40		None												
							12	AT	18	21	3	3¾	9¾		None												
							13	AP	18	21	3	100	260		None												
							14	AT	21	26	5	7½	37½		None												
							15	AP	21	26	5	100	500		None												
							16	AT	26	28	2	9½	16½	105¾	None												
							17	AP	26	28	2	100	220	2,060	None												
2	Bronchial asthma	Moderate	34	Negro	M.	48	1	AT	1	15	14	9	126	126	None	1	0	0	0	2	86	11,200	Not done			1	*Diplococcus pneumonia* (Type 25) (numerous)
							2	AP	1	9	8	180	1,410		None	42	0	0	0	42	90	8,800					
							3	OT	1	5	4	24	88		None												*Viridans streptococcus*
	Lobar pneumonia						4	OT	5	15	10	32	316		None											22	*Viridans streptococcus*
							5	OT	15	34	19	32	624		None												*Staphylococcus albus*
	Pulmonary cyst						6	AP	27	46	19	120	2,280	3,690	None												
							7	OT	35	40	5	32	160		None												
							8	OT	40	49	9	40	345	1,533	None												

3	Bronchial asthma Upper respiratory infection	Moderate	3	White	F.	8	1	AT	1	6	5	9	45	45	None	Not obtained				2	89	18,500	Not done			1	*Viridans streptococcus*
							2	AP	1	6	5	60	300	300	None					6	95	12,550					*Staphylococcus albus*
							3	OT	6	9	3	4	12	12	None												
4	Bronchial asthma Bronchopneumonia	Severe	32	Negro	M.	9	1	AT	1	8	7	9	63	63	None	1	2+	occ.	occ.	1	98	8,850	Not done				Not obtained
							2	AP	1	8	7	120	840	840	None	7	0	occ.	0	7	89	4,700					
							3	IMP	1	2	1	400	500	500	None												
							4	OT	1	5	4	24	88		None												
							5	OT	5	8	3	32	110		None												
							6	OT	9	10	1	32	48	246	None												
5	Bronchial asthma	Severe	48	White	M.	8	1	AT	2	6	4	9	37½	37½	None	4	0	0	0	3	111	12,450	Not done			1	*Neisseria catarrhalis*
							2	AP	2	6	4	120	500	500	None												
							3	IMPB	1	8	7	600	4,800	4,800	None											7	*Escherichia coli* (heavy growth)
							4	OT	8	9	1	16	16	16	None												*Gamma streptococcus* (few)
6	Bronchial asthma Hypertrophic maxillary sinusitis Antral polypi Chronic gingivitis Abraded teeth	Severe	56	White	M.	84	1	AT	1	11	10	9	88½		None	1	0	0	0	1	100	8,100	3	13	17	1	*Neisseria catarrhalis*
							2	AP	1	11	10	120	1,180		None	82	0	0	0	82	96	8,000	33	13	13		*Escherichia coli*
							3	OT	1	10	9	30	275		None												*Gamma streptococcus*
							4	OS	1	7	6	6	41		None												
							5	IMP	7	22	15	400	5,850		None												
							6	OT	10	17	7	40	260		None											7	*Escherichia coli* (pure culture)
							7	AT	11	13	2	7½	16½		None												
							8	AP	11	13	2	100	220	1,400	None											15	*Pseudomonas pyocyanea*
							9	OT	17	18	1	20	30		None												*Escherichia coli*
							10	OT	20	31	11	20	235	800	None												*Hemolytic staphylococcus albus*
							11	AT	20	22	2	18¾	37½		None												*Gamma streptococcus*
							12	AT	22	23	1	15	11¼		None											33	*Escherichia coli* (heavy growth)
							13	AT	23	28	5	36	9	162¾	None												*Hemolytic staphylococcus albus*
							14	OS	23	39	16	6	98	139	None												*Viridans streptococcus*
							15	IMP	39	64	25	400	10,050	15,900	None												
7	Bronchial asthma	Severe	37	Negro	F.	16	1	AT	1	8	7	18	123		None	3	0	occ.	0	3	100	7,300	4	19	32	1	*Escherichia coli* (pure culture; heavy growth)
							2	AP	1	8	7	120	820		None	10	0	occ.	0	10	92	7,800	7	17	18		
							3	OT	1	17	16	60	970	970	None											7	*Escherichia coli*
							4	IMP	1	11	10	400	3,900	3,900	None											12	*Escherichia coli*
							5	AT	10	11	1	9	10½		None												
							6	AP	10	11	1	120	140		None												
							7	AT	11	15	4	7½	28½	162	None												
							8	AP	11	15	4	100	280	1,340	None												
							9	OS	11	13	2	6	13		None												
							10	OS	13	17	4	5	21	34	None												

TABLE 1—*Continued*

CASE NO.	DIAGNOSIS	DEGREE OF SEVERITY	AGE IN YEARS	COLOR	SEX	DAYS OF HOSPITALIZATION	SERIES OF DRUG	FORM OF DRUG*	HOSPITAL DAY BEGUN	HOSPITAL DAY ENDED	DURATION OF THERAPY IN DAYS	DAILY DOSE*	TOTAL SERIES DOSE*	TOTAL DRUG DOSE*	UNTOWARD SYMPTOMS	LABORATORY											
																Urine				Hematology			Prothrombin Time in Seconds			Bateriology of Sputum	
																Hospital Day	Albumin	WBC	RBC	Hospital Day	% Hb	WBC	Hospital Day	Control	Patient	Hospital Day	Organisms
8	Bronchial asthma Pulmonary emphysema marked	Severe	42	White	F.	30	1	AT	1	5	4	9	37½		None	1	0	occ.	0	2	115	11,600	2	16	17	1	*Viridans streptococcus*
							2	AP	1	5	4	120	500		None	18	0	10	0	19	100	5,850					*Staphylococcus albus*
							3	IMP	1	10	9	400	3,600	3,600	None												*Neisseria catarrhalis*
							4	OT	1	8	7	20	130		None											8	*Neisseria catarrhalis*
							5	AT	5	31	26	7½	192	229½	None												*Escherichia coli*
							6	AP	5	12	7	100	720		None											11	*Escherichia coli* (heavy growth; pure culture)
							7	OT	8	18	10	30	320		None												
							8	OS	10	31	21	6	130	130	None											18	*Viridans streptococcus* (pure culture)
							9	AS	12	20	8	2	15.2		None												
							10	OT	18	31	13	40	500	950	None											25	*Gamma streptococcus* (few)
							11	AP	20	30	10	100	1,020	2,240	None												*Yeast cells, saccharomyces group* (moderate)
							12	AS	20	30	10	2	20.4	35.6	None												
9	Bronchial asthma	Severe	56	Negro	M.	10	1	AT	1	2	1	72	37½		None	3	0	0	0	3	92	7,500	2	14	16	2	*Neisseria catarrhalis*
							2	AP	1	11	10	120	1,160	1,160	None	9	0	occ.	0								*Viridans streptococcus*
							3	IMP	1	11	10	400	3,900	3,900	None												*Staphylococcus aureus*
							4	OT	1	11	10	60	585	585	None											9	*Escherichia coli* (pure culture; heavy growth)
							5	AT	2	11	9	9	82½	120	None												
							6	IMPB	11	11	1	300	300	300	None												
10	Bronchial asthma	Moderate	57	White	F.	10	1	AT	2	6	4	9	36		None	2	1+	6–8	0	2	80	8,400	Not done				Not obtained
							2	AP	2	6	4	120	480		None												
							3	IMP	2	11	9	400	3,550	3,550	None												
							4	OT	4	8	4	30	120		None												
							5	AT	6	8	2	7½	12		None												
							6	AP	6	8	2	100	160		None												
							7	OT	8	11	3	40	110	230	None												
							8	AT	9	11	2	7½	20½	68½	None												
							9	AP	9	11	2	100	260	900	None												

*A: Aerosol.
O: Oral.
IM: Intramuscular.
T: Theoglycinate; doses are in grains.
P: Penicillin; doses are in thousands of units.
S: Sulfadiazine; doses are in grams.
PB: Penicillin in beeswax; doses are in thousands of units.

COMMENTS

Patient #1. See *Case No. 1* and *Discussion:* The patient made no complaint of nausea until the fifth hospital day, because his asthma was so much improved that he was afraid any change of therapy would result in an exacerbation of his dyspnea. At this time, he admitted that he had had constant nausea since two days preceding admission. Twelve hours after the decrease in the dose of oral theoglycinate from sixty-four to twenty-four grains daily, he became quite hungry, and had no further nausea or anorexia. Skin tests to common inhalants were negative, a further indication that his asthma was on an infectious basis.

Patient #2. See *Case No. 2* and *Discussion:* Negative epidermal sensitivity tests to common inhalants confirmed the impression of an infectious basis for the asthma.

Patient #3. See *Case No. 3* and *Discussion:* Skin tests were positive to house dust and fungi, indicating that both extrinsic and intrinsic factors were the basis for the asthma.

Patient #4. This 32 year old negro severe asthmatic male was admitted March 9, 1947, in moderate respiratory distress. His asthma, which had been present constantly for four days, had been unrelieved by the usual parenteral therapy. He gave a history of a mild chill and fever one day preceding admission. When seen in the accident room at this time, his temperature was 101°F. with typical signs of asthma and signs of bronchial pneumonia at both bases (not confirmed by chest roentgenography). He was started on penicillin intramuscularly, 50,000 units every three hours, but his dyspnea continued after twenty-four hours despite a drop in temperature to 99.2°F. Therefore, he was admitted for theoglycinate and penicillin aerosol therapy. After two treatments there was appreciable clinical relief from the dyspnea. At the end of twenty-four hours, at which time the intramuscular penicillin was stopped, he was no longer dyspneic. His chest had become clear on auscultation by the fourth day of aerosol treatment and remained so. No further asthmatic attacks occurred during his hospitalization. He was discharged March 18, 1947, taking eight grains of theoglycinate orally four times daily. However, he has not been well in the following seven months. Evidence of a sinus infection has persisted, and asthmatic attacks have recurred frequently. Some have responded favorably to extra ten to fifteen grain doses of oral theoglycinate in addition to a daily dose which has been increased to sixty grains. The remainder of the attacks have required parental therapy, and usually have responded readily.

Patient #5. This 47 year old white severe asthmatic male was admitted in acute respiratory distress on May 6, 1947. He had had constant asthma for three days without relief from rigorous parenteral therapy. He had been hospitalized three weeks previously and had received theoglycinate and penicillin aerosol therapy for a similar episode, present for one day and unresponsive to the usual parenteral therapy. On that occasion his dyspnea had disappeared after twenty-four hours with aerosol treatment, and he had been asymptomatic until the present asthmatic attack. Immediately following admission, he received transitory relief from two mgm. of butanefrine intravenously, but then became refractory to further parenteral therapy. His dyspnea grew progressively more severe, and after twenty-four hours his condition had become quite critical, with marked apprehension and exhaustion, and persistent cyanosis despite continuous oxygen. The breath sounds at both bases were just audible on auscultation. Eight hours after the initiation of aerosol therapy, while receiving the third treatment with theoglycinate aerosol, the patient had a violent coughing paroxysm and brought up about an ounce of thick tenaceous sputum. He immediately showed a marked clinical relief from his dyspnea, with disappearance of his cyanosis. An ausculatory examination of his chest, begun just prior to the onset of the coughing paroxysm with no change in findings from those eight hours before, revealed a sudden marked increase in the breath sounds at the bases posteriorly. The patient continued to have moderate dyspnea throughout the next day and night, with no other notable change in his condition, except that he was able to sleep. He was quite comfortable, however, by the third morning of hospitalization, thirty-six hours after initiation of the aerosol therapy, but although there was no outward evidence of asthma, signs persisted on auscultation. These signs had disappeared by the fourth day of treatment, and except for two minor episodes of asthma readily controlled with butanefrine, he had no further severe asthmatic attacks. He was discharged on May 14, 1947, at which time signs of chronic bronchitis had returned on auscultation. He was given butanefrine for self-administration and four grains of theoglycinate four times daily orally. He has required hospitalization two times in the five months since, receiving the same type of treatment in addition to surgical removal of recurring nasal polypi. On both occasions he again responded favorably to the treatment.

Patient #6. This patient was admitted in no respiratory distress, but gave a history of daily asthma for the preceding four months with only transitory relief of symptoms. He was now unresponsive to the usual parenteral therapy. He did well for the first ten days and then began having repeated asthmatic attacks, which responded well, however, to parenteral therapy. On the fourteenth hospital day he was given a therapeutic general anesthetic without improvement, and his attacks continued to grow more numerous. On the twenty-ninth hospital day, a Caldwell-Luc was performed on the left maxillary antrum. Numerous, obviously infected polypi were removed. Following this procedure, the asthmatic attacks practically ceased. On the forty-third hospital day, a Caldwell-Luc was performed on the right maxillary antrum. The asthmatic attacks continued to be minimal, and on the fifty-second hospital day, two of his six remaining teeth were removed. On the fifty-ninth hospital day, the other four were removed. During the rest of his hospital stay, prolonged because of unsatisfactory home care, asthmatic attacks were rare. Theoglycinate was stopped because of complaints of abdominal distress, but this continued throughout his hospital stay, and appeared to be the result of prolonged bed rest.

Patient #7. This patient was admitted in severe respiratory distress, having been in status asthmaticus for the preceding two weeks and receiving no benefit from parenteral therapy and only minimal relief from theoglycinate and penicillin aerosol in the accident room for the forty-eight hours preceding hospitalization. Four hours after admission, the patient was quite comfortable. The chest was clear on auscultation by the fifth hospital day. A roentgen ray of the chest showed infiltration at the right apex on admission. After two weeks, the chest roentgen ray was negative. The anterior tip of the right middle turbinate was removed surgically on the eighth hospital day, because of polypoid degeneration. Two carious teeth were also removed on the thirteenth hospital day. The patient did well for three weeks, when all theoglycinate was stopped for forty-eight hours because of obscure gynecologic complaints. She was readmitted in status asthmaticus with a similar satisfactory response to treatment with theoglycinate and penicillin aerosol. The gynecologic complaints were later proved to be the result of a pelvic abscess.

Patient #8. This patient was admitted with moderate dyspnea, giving a history of constant asthma for the preceding ten days, receiving only transitory relief from parenteral therapy. She was comfortable clinically after twenty-four hours, and except for occasional mild episodes of dyspnea readily controlled by two mgm. of butanefrine intramuscularly, remained so. Signs of bronchial asthma subsided gradually over a four day period, but signs of chronic bronchitis persisted. The patient was dyspneic at bed rest, because of marked pulmonary emphysema and a reduced vital capacity. Her dyspnea gradually became less marked, and she was able to resume light activity without her previous discomfort. *Escherichia coli* was present in the sputum on the eighth day of penicillin aerosol therapy, and when a pure culture of this organism was reported on the eleventh day, the patient was put on sulfadiazine aerosol. After receiving this treatment for seven days, a pure culture of *Streptococcus viridans* occurred. The patient was then put on combined penicillin and sulfadiazine aerosol for ten days, with gamma streptococcus and yeast cells reported on the fifth day of treatment. Improvement continued two weeks after discharge from the hospital, when there was an exacerbation of her asthmatic symptoms as a result of a fresh upper respiratory infection. This was readily controlled with theoglycinate and penicillin aerosol therapy.

Patient #9. This patient was admitted from the dispensary, irrational and practically moribund, with circulatory failure and the history of a mild convulsive seizure immediately preceding admission. He had been in status asthmaticus for two days following an acute upper respiratory infection. He improved gradually with continuous theoglycinate aerosol to such an extent that this could be discontinued after twelve and one-half hours. Twenty-four hours after admission, he was completely comfortable at bed rest. His mental status had improved, and he was able to give a coherent history. His chest was clear of signs of asthma on auscultation by the end of the fourth hospital day. He had no further asthmatic attacks during his hospitalization.

Patient #10. This patient was admitted in moderate respiratory distress, giving a history of constant asthma for three weeks, unresponsive to the usual parenteral therapy. Her asthma occurred following the onset of an upper respiratory infection. There was marked clinical improvement after twenty-four hours of treatment with theoglycinate and penicillin aerosol. Her symptoms of dyspnea gradually subsided until the eighth day of hospitalization, when she received no aerosol treatments for thirty-six hours as a result of an error in the interpretation of orders. She was promptly relieved by the first subsequent aerosol treatment, and she remained asymptomatic throughout the remainder of her hospitalization.

considered moderate exhibited orthopnea, but were able to breathe in a reclining position with the head of the bed elevated. They were acyanotic, with no marked use of the accessory muscles of respiration, and the quality of the breath sounds was unimpaired. No patients were hospitalized who exhibited mild asthmatic attacks. A history of previous severe attacks, controlled with difficulty, was considered of great significance in the evaluation of the severity of the attacks under consideration.

Untoward Effects: In only one case was treatment stopped because of undesirable effects. This patient exhibited bronchorrhea. There was no oral discomfort, as has been noted when penicillin was administered by a nebulizer with a mouthpiece. There was no evidence of nasal or eye irritation. No effects were noted on the blood count or the urine. An occasional case experienced slight or moderate nausea following the use of oral theoglycinate in large doses, but this was readily controlled by decreasing the dose. A few complained of nausea when aerosol was given immediately before or after meals, but a change in the timing of the treatment prevented the recurrence of this complaint.

TABLE II

Tabulation of Duration and Dose of Theoglycinate and Antibiotics Administered to Ten Patients Hospitalized with Bronchial Asthma

NUMBER OF PATIENTS RECEIVING DRUG	FORM OF DRUG*	DURATION OF THERAPY IN DAYS			DOSE*				
					Total	Daily		Total	
		Total	Variation	Average		Variation	Average	Variation	Average
10	AT	125.4	4–30	12.54	1,119.5	3.75–42	8.9	37.5–229.5	112
10	AP	121.4	4–27	12.14	14,430	60–200	118.9	300–3,690	1,443
7	IMP	88	1–25	12.57	33,480	240–400	380.5	500–15,900	4,782.9
10	OT	177	1–47	17.7	6,124	4–60	34.6	12–1,533	612.4
3	OS	49	6–32	16.33	333	5–6	6.8	34–139	111
1	AS	18	18	18	35.6	2	2	35.6	35.6
2	IMPB	8	1–7	4	5,100	300–600	627.5	300–4,800	2,550

* A: Aerosol.
IM: Intramuscular.
O: Oral.
T: Theoglycinate; doses are in grains.
P: Penicillin; doses are in thousands of units.
S: Sulfadiazine; doses are in grams.
PB: Penicillin in beeswax; doses are in thousands of units.

OTHER MEASURES

The conventional measures previously mentioned for the control of status asthmaticus were used freely in the cases under consideration. All of them gave temporary relief in certain cases, but this was almost never sufficiently sustained. The authors have been depending more and more upon the aerosol routine outlined herein, because of its effectiveness, and not only its seeming lack of serious toxicity, but its lack of even minor reactions of an unpleasant or disturbing nature.

SUMMARY

A method is presented for the control of attacks of severe bronchial asthma with theoglycinate and penicillin aerosol. This has proved to be a safe and effectual method of control. It has been used in 33 cases of bronchial asthma, and in the authors' opinion, is superior to other methods of treatment known to them.

CONCLUSION

Treatment of severe bronchial asthma by the aerosolization of sodium theophylline glycinate solution, followed by a properly selected antibiotic agent, is a safe and effective procedure. In the authors' hands it has been frequently successful when conventional methods have been ineffectual.

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CURRENT TRENDS IN SPECIFIC THERAPY OF BACTERIAL MENINGITIS* †

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Within recent years three potent, new, therapeutic agents have become available for the treatment of bacterial meningitis. In order of appearance these are the sulfonamides, penicillin and streptomycin. Mortality figures, already reduced by the use of specific antisera, have been markedly lowered. Although indications for the use of these drugs are well known, their use in a specific case of bacterial meningitis is often subject to improvement. This paper represents a study of 95 cases of bacterial meningitis admitted to the University of Maryland Hospital since early 1945 (Table I), along with a review of the current medical literature. It is an attempt to determine the best therapeutic regime in each specific type of bacterial invasion of the meninges.

It is surprising that in this series, meningococcus meningitis ranks only third in number. Possibly many cases of meningococcal infections are treated with specific chemotherapy before clinical meningeal invasion occurs. The relative incidence of influenzal meningitis in this series is the highest reported in the American literature.

The importance of early diagnosis cannot be overemphasized, especially in the fulminating case. In children, and occasionally in adults, the classic signs of meningeal irritation may be minimal. Concurrent otitis, mastoiditis, sinusitis, pneumonia or septicemia may confuse or mask the general aspects. An alteration in the state of consciousness, a headache, or a relative bradycardia may be the only clue. These signs may suggest a lumbar puncture; and whenever this procedure is considered, it should be done. This is especially true if the patient has an acute severe febrile illness of an obscure nature. Sufficient spinal fluid should be withdrawn for cytologic, bacteriologic and biochemical study. If cloudy or purulent spinal fluid is found, the diagnosis of bacterial meningitis must be assumed; and in order to save valuable time, intravenous administration of five grams of sodium sulfadiazine in 500 cc. of isotonic saline should be started immediately. This initial dose should be followed by maintenance doses of two grams every eight hours parenterally or one gram every four hours orally if the patient can retain the drug. Subsequent sulfadiazine dosage should depend both on clinical response and on blood levels of the drug.

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† Read before a staff meeting of the University Hospital, Baltimore, Maryland on December 18, 1948.

Stained smears of the spinal fluid sediment should be made immediately, and a careful search carried out for the specific invading organism. In a high percentage of cases a tentative specific diagnosis may thus be made. This is especially true in the influenzal and pneumococcal varieties (27). If the invading organism can be identified by a smear, immediate specific therapy may be instituted as described hereafter. If the organism cannot be identified in smears, sulfadiazine administration is continued, awaiting the results of cultures.

TABLE I

TYPE	NUMBER	DIED	PER CENT	CORRECTED PER CENT*
Influenzal	23	5	22	22
Tuberculous	23	23	100	100
Meningococcic	21	3	14	5
Pneumococcic	20	7	35	28
E. coli	3	0	0	0
Misc. and undetermined	5	2	40	40

* After subtraction of cases dying in less than twenty-four hours.

TABLE II

AGE	NO. OF CASES	MORTALITY
Up to 6 months	4	75%
6 months to 2 years	11	9%
2 years to 12 years	2	0%
12 years to 18 years	2	0%
Above 18 years	4	25%

INFLUENZAL MENINGITIS

Influenzal meningitis is most common in children under two years of age. There are few case reports of this disease in adults. Table II shows the mortality according to age distribution in this series.

These mortality figures of patients under 6 months of age compare favorably with those of other authors (5, 8). The only death in a patient above two years of age was that of a 57 year old male with a complicating endocarditis, who died twelve days after admission. In 58 per cent of the cases in this series there was an associated bacteremia, as shown by positive blood cultures, but the above was the only case of associated endocarditis.

There are three effective antibacterial agents available for the treatment of meningitis as a result of type b *Hemophilus influenzae:* sulfadiazine, type b Hemophilus influenze rabbit antiserum, and streptomycin. Each of these agents exerts its effect through a different mechanism. Hence the problem facing the internist today is the choice of the drug or drugs to be used and of the route of administration. The four most useful therapeutic regimes will be discussed briefly.

Sulfadiazine and antiserum: In this series, ten cases were treated with sulfadiazine and type b Hemophilus influenzal antiserum with a mortality rate of 30 per cent. This is perhaps the oldest effective therapeutic combination and good results are cited by several clinics (1, 5, 18, 19). Antiserum is effective only against type b. However, all cases in this series and 95 per cent of the cases reported by Alexander (1), are caused by type b.

The severity of the infection varies inversely with the spinal fluid sugar concentration. The following dosage schedule recommended by Alexander (1) is based on this concept.

Schedule of dosage based on spinal fluid sugar

SPINAL FLUID SUGAR (MG. PER CENT)	MG. OF ANTIBODY NITROGEN INDICATED
15	100
15–25	75
25–40	50
over 40	25

The necessary quantity of antiserum is diluted in 10 cc. of isotonic saline per kilogram of body weight and given intravenously over a two hour period. Additional antiserum administration will depend upon clinical response and the presence or absence of demonstrable antibodies in the patient's serum. This may be determined by mixing the patient's serum and a suspension of type b. Hemophilus influenzal organisms on a glass slide; capsular swelling will indicate adequate serum antibody concentration. This test should be performed within twenty-four hours after antiserum administration at least every other day during the first week. If the clinical response is not satisfactory or if the serum does not produce capsular swelling, antiserum administration should be repeated (19, 20). Sulfadiazine administration should be continued, according to the schedule previously outlined, until there is complete clinical recovery and the spinal fluid findings have returned to normal.

Streptomycin: Only three cases in this series were treated with streptomycin alone. All of these recovered. Alexander, Leidy, Rake and Donovich (2) successfully treated several cases with streptomycin, but they had two unsuccessful cases in which the strains showed decided resistance to streptomycin. Birmingham, Hayes and Smith (6) reported eight cases treated with streptomycin, either at the beginning of therapy or following unsuccessful treatment with other therapeutic agents. They had three fatalities. Paine and Finland (19) believe it is the most effective means for treating the disease.

Streptomycin possesses certain advantages as compared to sulfadiazine-antiserum therapy. First, the relatively high cost of antiserum and the difficulty occasionally encountered in its procurement would be abolished. Second, the danger of immediate or late reaction to heterologous serum would be eliminated. Third, streptomycin has been shown to be effective against all six types of encapsulated *H. influenzae* as well as the non-typable varieties which occasionally cause acute bacterial endocarditis in adults (1). Type b. Hemophilus influenzal antiserum, the only therapeutic serum available at present, is effective only against type b.

Intrathecal injection of 50–100 mg. of streptomycin once daily, with concurrent intramuscular injections of .25 to 2 grams daily in divided doses, is recommended in infants and children (19). Higher intramuscular dosage is used in adults. In this series toxic reactions were minimal, and negative blood and spinal fluid cultures were obtained in twenty-four to forty-eight hours.

Large doses of streptomycin do not prevent the establishment of a new infection by organisms not susceptible to the drug. This is pointed out by Weinstein (31), who reported 2 patients with influenzal meningitis who developed staphylococcus aureus meningitis and bronchopneumonia with bacteremia while receiving streptomycin. It is advisable to obtain frequent blood, throat and spinal fluid cultures so as to detect promptly the presence of a secondary invading organism which may require a change of therapy for its control.

Streptomycin-sulfadiazine: Ten cases reported here were treated with streptomycin and sulfadiazine with only 2 fatalities. The authors believe this represents the best therapeutic combination for influenzal meningitis. Cases of influenzal meningitis have been reported in which the organisms developed resistance to streptomycin. This is believed to be the result of the existence of a few highly resistant variants in any given bacterial strain (2, 6). Concurrent administration of sulfadiazine should inhibit these resistant variants and decrease the incidence of so-called fastness to streptomycin.

In three cases listed as influenzal meningitis in this series, there were other organisms found in the initial spinal fluid culture. The organisms found, along with hemophilus influenzae, were pneumococcus—type 6; pneumococcus—type 15; and the meningococcus. Mixed meningeal infections are rare (21, 24), and many cases may result from a contamination of cultures. Toomy and Roach (28) found a case of mixed penumococcal and H. influenza meningitis. Gaffney (13) reported a case of mixed meningitis with four spinal fluid cultures showing *H. influenzae* and two cultures growing out both *H. influenzae* and pneumococci. Other mixtures have been reported (14). The association of organisms may be found initially, as occurred in the three cases in this series, or the second organism may appear later. Rosenblum, Pearlman and Stanley (24) state that most secondary invading organisms appear between the seventh and eleventh day.

Because of the possibility of secondary invading organisms, the occurrence of drug fastness, and the occasional presence of mixed infections, the authors believe sulfadiazine administration is indicated in addition to streptomycin.

Streptomycin-sulfadiazine-antiserum: A combination of streptomycin, sulfadiazine, and antiserum should be used in severe infections occurring in children under 6 months of age. The mortality rate in this age group is still very high. The use of these three agents, working through different mechanisms, may be expected to reduce the risk of failure.

TUBERCULOUS MENINGITIS

All of the cases of tuberculous meningitis treated in this hospital ended fatally. Owing to the high cost of prolonged streptomycin therapy, this drug has not been available here for a clinical trial in tuberculous meningitis. Only one case has been given streptomycin; and because there is still a paucity of literature on streptomycin therapy in this condition, the following case is presented:

(Chart #21892). A 22 year old white male was admitted to Dr. T. N. Carey's private service June 20, 1946 because of a headache and fever of two weeks duration. His past history revealed diabetes mellitus for six years and known pulmonary tuberculosis for two months. A physical examination on admission revealed moderate nuchal rigidity a positive Kernig's sign, and fine moist rales throughout his lung fields. Chest roentgenographs showed bilateral pulmonary infiltration with probable cavitation in the upper lobe of the left lung. The initial spinal fluid examination showed a pressure of 198 mm. of water, pandy test of one plus, sugar 88 mg. per cent, protein 158 mg. per cent, and chlorides 430 mg. per cent. The fluid contained 175 white blood cells of which 80 per cent were lymphocytes. Stained smears did not reveal any organisms. A guinea pig was innoculated and therapy was started two days after admission. He was given between 200 and 400 mg. of streptomycin intramuscularly every three hours for two months for a total of 120 grams. He was given intrathecal streptomycin in daily dosage between 25 and 50 mg. for a little over two months. After four days there was a definite improvement in his condition, but his temperature continued to spike daily to 103 F. for six weeks. He then became afebrile and seemed much improved for ten days. Following this, the temperature again rose and clinical signs of meningitis became much more marked. His course was steadily downhill until his death on November 7, 1946. An autopsy showed tuberculous invasion of the meninges and the lungs.

Arrested cases of tuberculous meningitis have been reported (9, 15, 17, 20) but a careful study of all streptomycin treated cases will have to be made in order to determine the optimum daily dosage and duration of treatment. Apparently prolonged streptomycin administration offers some hope for victims of this otherwise fatal disease.

MENINGOCOCCIC MENINGITIS

Present day therapy of meningococcic meningitis has evolved itself into a choice of two potent antibiotics, sulfadiazine and penicillin, or a combination of the two.

Although sulfadiazine is considered the drug of choice (10, 20, 33), some reports of penicillin therapy alone compare favorably with sulfonamide treated cases (12). Best results may be expected when these two drugs are given simultaneously (7). Some reports indicate that therapeutic levels of penicillin may be attained in the spinal fluid following intramuscular therapy alone in large doses (22, 23), and it is believed that the average case will respond favorably to this regime. As intrathecal penicillin may result in toxic neurologic complications (11, 29) and as it may incite a polymorphonuclear pleocytosis (12), its routine use is not advocated in meningococcic meningitis.

In the series of 21 cases herewith reported, 11 received a combination of sulfadiazine and penicillin, but in none of these was the intrathecal route used. One case responded to intramuscular penicillin alone. Nine cases treated with sulfadiazine alone recovered, while the one fatality was in the sulfadiazine-penicillin group—a 70 year old Negress seen late in her illness following two days of coma. She failed to respond to adequate sulfadiazine administration and 40,000 units of penicillin intramuscularly every three hours. In this case, intrathecal penicillin might have been beneficial.

The use of specific antisera, when available, may make a slight improvement in the mortality rate among poor risk patients, but in general, its use is considered unnecessary.

PNEUMOCOCCAL MENINGITIS

A high percentage of the cases of pneumococcal meningitis are preceded by otitis media, sinusitis, mastoiditis, or pneumonia (4, 32). In 50 per cent of the cases of this series there was a history of a primary focus of infection. Prompt vigorous therapy should be directed toward the primary focus as well as the meningeal infection. A myringotomy or mastoid operation may be necessary while the patient is very ill. Failure to operate may significantly alter the prognosis (30).

Recurrent attacks of pneumococcal meningitis may occur in cases of cerebrospinal rhinorrhea. To illustrate this relationship the following case is cited:

W. K., a white male age 6 years, was first admitted to the hospital in 1941 for a plastic repair of his frontal bone following a kick by a mule. In spite of repeated operations he had a persistent cerebrospinal rhinorrhea. He was readmitted on four occasions because of pneumococcal meningitis. On each admission the diagnosis was made by spinal fluid culture. The first two infections were treated with sulfonamides alone while the others, both in 1945, were given sulfadiazine plus intrathecal and intramuscular penicillin. Following his last infection the defect was successfully repaired surgically.

Table III outlines the therapy and the results obtained in the authors series of pneumonacoccus meningitis.

Two of the deaths occurred in less than twenty-four hours. Another was a 75 year old patient who never fully recovered from the effects of the disease and died one month after his meningeal infection had cleared.

TABLE III

NO.	SEX	AGE (YEARS)	INTRAMUSCULAR PENICILLIN	INTRATHECAL PENICILLIN	SULFADIAZINE	SERUM	RESULT
1	M	70	+		+		D
2	F	64	+	+	+	+	D
3	F	2	+		+		D
4	F	8 months	+	+	+		D
5	M	10	+	+	+		C
6	M	53	+		+		D
7	M	16	+		+		D
8	M	63	+		+		C
9	M	10	+	+	+		C
10	F	10	+	+		+	C
11	M	7 months	+	+	+		C
12	F	1	+		+		C
13	M	1	+	+	+		C
14	M	50	+		+	+	C
15	M	45	+	+	+		C
16	M	35	+		+		C
17	M	28	+	+	+		C
18	F	10 months	+	+	+		C
19	M	52	+	+	+		C
20	M	75	+	+	+		D

Sulfadiazine plus penicillin was used in all but one of these cases. The latter patient was successfully treated with intramuscular plus intrathecal penicillin and pneumococcus antiserum. Intrathecal penicillin was used in twelve cases. Only three of these patients died. One died in less than twenty-four hours and another after one and one-half months. Four deaths occured in the twelve patients who did not receive intrathecal penicillin.

Penicillin, 50,000 units every three hours, should be given in addition to the administration of sulfadiazine as outlined previously. Because of a marked tendency to relapse (20), this therapy should be continued for at least seven days after there is both clinical and laboratory evidence of recovery. The intrathecal administration of penicillin is indicated (4, 25), daily for the first few days. This ensures an early adequate concentration in the spinal fluid. The daily administration of 10,000 units in 10 cc. of saline for three or four days is usually adequate, but it should be continued until there is a clinical response.

Pneumococcus antiserum may be of some value, but its use should be reserved for those cases that do not respond to the above regime.

ESCHERICHIA COLI MENINGITIS

Meningitis as a result of Escherichia coli is a rare and formerly a highly fatal disease (3, 26). Three successfully treated cases are included in this series. Two were adults and the third was a two day old infant. These cases are to be reported in detail in another article.

This type of meningitis may follow penetrating wounds of the meninges or be secondary to a distant focus of E. coli infection (20). Early surgical treatment may be indicated in addition to chemotherapy.

Streptomycin is the most valuable therapeutic agent and the response may be quite dramatic (16). Concurrent administration of sulfadiazine may be beneficial.

MISCELLANEOUS AND UNDETERMINED

This group in the authors series includes one case of meningitis caused by streptococcus viridans and a second caused by an organism of the proteus group. Two fatal cases occurring in infants were of undetermined etiology. The therapy in each of these cases varied considerably. Staphylococcic and streptococcic meningitis can be effectively treated by sulfadiazine plus intramuscular and intrathecal penicillin (20, 32). If the invader is one of the gram-negative organisms, sulfadiazine and streptomycin may be given according to the procedure outlined for influenzal meningitis.

SUMMARY

Successful specific treatment of bacterial meningitis is dependent upon early diagnosis of the disease and identification of the causative organism.

Classic signs of meningitis are frequently of late appearance. Minor evidences of central nervous system involvement are an indication for diagnostic lumbar puncture in any severe obscure febrile illness.

In the presence of purulent spinal fluid, the immediate administration of sodium sulfadiazine parenterally is advocated. Further specific therapy will vary according to the type of organism identified by smear and culture.

The use of sulfonamides, penicillin, and streptomycin and specific antisera in meningitis of influenzal, meningococcic, pneumococcic, E. coli, and tuberculous etiology is discused on the basis of an analysis of 95 cases and a review of the recent medical literature.

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LIPOSARCOMA: A REPORT OF TWO CASES*†

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Since Virchow's original description of liposarcoma in 1857, recognition of the tumor has been progressively more widespread. The number of cases reported in the literature has been particularly abundant during the past two decades. The greatest single series was that published by Stout (20) in 1944, where he provided an excellent analysis of 41 cases.

This paper will review briefly the salient pathologic features of the liposarcoma as recognized to date, and present case histories. No attempt will be made to discuss the clinical problems arising from the tumor.

INCIDENCE

In a survey of the literature prior to 1939, Lingley (11) found an equal incidence in the two sexes. Murray and Stout (16) on the other hand reported twice as many cases in males as in females. However, studies of the published case reports seem confirmatory of an equal sex distribution.

The age group in which the liposarcoma appears is highly variable. Among Stout's (20) 41 cases, the mean age was 53 years, with 60 per cent beyond the age of 40. Murray (16) reported an average age of 56.5 years, with a range of from 28 to 82 years. A mean age of 50 years, with a range of 8 to 69 years, was arrived at from a review of the literature. One case of a liposarcoma in the newborn (5) was not included among these statistics.

Seventy per cent of the tumors were found in persons between 20 and 60 years of age, with the greatest number of these occurring during the fourth and fifth decades. Ten per cent occurred prior to the age of 20 and twenty per cent occurred after the age of 60. Thus liposarcoma is seen to be no respector of either age or sex.

SITES OF OCCURRENCE

The potential sites for development of the liposarcoma are many. However, Ackerman (1) lists as the most frequently involved areas: the popliteal

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spaces, about the buttox, and within the retroperitoneal tissues. Lingley (11) believes the soft tissues of the lower extremities to be the predominant site of occurrence.

Upon studying the reports in the American journals of the past three decades, it was discovered that about equal numbers of cases were reported with involvement of the soft tissues of the legs or of the retroperitoneal space (24 per cent each). The latter group was increased somewhat if one included those tumors reputedly arising within the kidney.

The remainder of the cases included a multiplicity of sites of origin, including, the femur (2, 18); the axilla (5); the greater omentum (14); the chest wall (15); the scapular region (15); the mediastinum, lung, or pleura (17, 23); the pelvic bones (18); the vulva (21); the popliteal space (23); and those of multiple origin (1).

One feature common to all of these neoplasms, irrespective of the site of development, was the tendency to recur following excision. This has been stressed by Ackerman (1), Duffy (2), Moreland (15), Lingley (11), and Murray (16).

Metastasis has not been reported as frequently as have regional recurrences, although, Stout (16) found twenty per cent of his series to have metastatic lesions. The most commonly mentioned sites of metastasis were the regional lymph nodes, the lungs, the liver, the bones, the spleen, and the heart and pericardium.

Mention should be made of the very interesting syndrome discussed by Fish (6) and Froug (8). This consisted of liposarcoma of the kidney with an associated tuberous sclerosis, and in other instances with an adenoma sebaceum of the face.

CHARACTERISTICS

In 1936 Shaw (19) listed the characteristic features of liposarcoma. These have undergone no important revisions since that time, and are briefly as follows: (1) It occurs in adults of both sexes; (2) Growth is slow for many years, with a sudden acceleration in the rate of development; (3) The encapsulation and ready removability do not suggest malignancy; (4) Local recurrence is usual and may frequently be repeated; (5) Death follows metastasis; (6) The histologic structure is variable.

Moreland (15) emphasized the final point above by pointing out the danger of confusion of the histologic structure with that of fibrosarcoma and that of xanthoma. In one of the cases reported in this paper there was for a period a tendency to confuse the tumor with one of fibroblastic origin, but finally this error was eliminated. Nevertheless, it is a pit into which the microscopist may fall if he is not constantly cognizant of the existence of this malignancy.

HISTOPATHOLOGY

The typical cell of normal fat is a large vacuolated cell surrounded by a membrane which in many ways resembles elastic fibers. The nucleus is small, eccentrically placed, and is surrounded by a small amount of granular cytoplasm. Golgi-apparatus has at times been identified by employment of special histologic techniques. Groups of these cells are found to be bound together by a fibrous tissue stroma.

Adult fat has been divided into two types dependent upon the color; brown or white. Liposarcoma has been found to develop from both types. The brown fat has a rather limited distribution within the mediastinum, perirenal areas, axillae, and joints. The white fat is much more abundant and comprises the remainder of the fat within the body.

In 1940 Wells (22) presented an exhaustive study of normal and abnormal adipose tissue. He contended that fat could be considered as a part of the reticulo-endothelial system inasmuch as fat arises from perivascular mesenchymal cells closely related to the reticulum. Hence, the fat arises from the same anlage as the lymph nodes. This he further substantiated by the reciprocal replacement of fat by lymphoid tissue and conversely the replacement of lymphoid tissue by fat.

Ewing (3) has further noted the close relationship between liposarcoma and myxosarcoma. He called attention to the fact that myxocarcoma, especially in the early stages of development, exhibits first a great overgrowth of blood vessels about which fat cells develop. The cells, lipoblasts and steatoblasts, are derived from the adventitia of the blood vessels and form a structure which in some respects resembles liver tissue. This configuration may be frequently seen in cases of cellular liposarcoma.

Ewing (3, 4) also divided liposarcomas into two primary types. The adult type is similar to the growth of fat cells which one frequently observes in association with chronic inflammatory processes. The cells are of the type seen in adult fat, with changes at first occurring within the cells and later becoming diffuse and of a variably malignant appearance. There is no primary growth of blood vessels and no tendency to form mucus.

The embryonal form arises with capillary proliferation as does the embryonal fat. The immature fat cells arise from the adventitial mesothelium, but instead of forming adult fat they form embryonal fat and/or mucus.

The latter form was designated as intermuscular myxoliposarcoma by Ewing, because of its anatomic relationships.

Grossly, many of the tumors are orange, yellow, or brown, especially those arising from the brown fat. These are highly cellular and contain little recognizable fat. Other liposarcomas may be yellow because of the presence of xanthoma cells. In many, multinucleated giant forms are present.

It should be stressed that the microscopic aspect is variable. The structure is dependent upon many factors, including histogenesis and location. Extreme care must be taken to differentiate the tumor from other cellular sarcomas.

Case Report I: The patient was a fairly well developed 58 year old white female, admitted to the University Hospital, Baltimore, Maryland on July 7, 1942. She had noticed an increase in her waistline and a gradual development of a lump on the right side of her abdomen for a period of one and one-half years prior to admission. Her past and family history were non-contributory.

A physical examination showed that the patient was in no apparent distress. The only significant finding was the abdominal mass on the right side. It extended from the costal margin to the anterior-superior spine and from the anterior axillary line to the lateral border of the left rectus muscle. It was movable and many lobulations were felt. The kidney could be felt independently of the mass, and it was thought that the tumor was not of renal origin.

A roentgenologic examination of the abdomen showed the soft tissue density in the right abdomen to be greater than normal. Following the intravenous injection of diodrast, the renal pelvis and calices were fairly well filled, and the right kidney was displaced downward. A barium enema was negative.

Laboratory blood studies showed 13.1 grams hemoglobin, 7,700 white blood cells with a normal differential, and a sedimentation rate of 41 mm. The non-protein nitrogen was 32 mgm. per 100 cc., and the blood sugar 78 mgm. per 100 cc. The urine showed no abnormalities, and the serologic tests for syphilis were negative.

Under general anesthesia, a long lower right rectus incision was made. The cecum and ascending colon were found pushed to the left and a large retro-peritoneal mass the size of a football was completely surrounding the right kidney. The mass was freed and care taken not to injure the kidney or ureter. A fair amount of bleeding was encountered.

The patient made an uneventful recovery and was discharged August 10, 1942 in good condition.

PATHOLOGIC DATA

Gross: The specimen consisted of three masses of doughy yellowish tissue which measured 11 x 6 x 14 cm., 10 x 12 x 4 cm., and 5 x 6 x 4 cm., respectively. On section the largest mass showed fatty tissue somewhat firmer than usual and containing a few small areas of gelatinous material. The medium and the smaller mass were well encapsulated, pale, firm, and showing marked increase of fibrous-like trabeculations through their substance.

Microscopic description: Numerous sections studied showed identical aspects except for a few areas of the largest mass where some normal adult type fat cells were present. The sections showed a highly cellular structure. The cells were polyhedral and contained as many as two or three nuclei. Some were elongated and resembled fibrous tissue, but showed many fatty droplets when stained with Sudan III. Mitotic forms were also found.

Readmission: The patient was readmitted on January 4, 1943 because of recurrence of a tumor at the original site. At this admission the patient appeared in good health. The incision was well healed. Under this incision a large mass was easily palpated that extended from the right side of the abomen across the midline and from the costal margin down to the umbilicus.

The patient was again prepared for surgery. Under general anesthesia an incision was made through an old right rectus scar, and after considerable difficulty the peritoneal cavity was opened. Many adhesions were encountered during this process. The mass was readily found and dissected free from the ascending colon after the lateral gutter had been opened. It was also dissected free on its upper aspects from the duodenum and pancreas. When the lower pole was dissected free the ureter was found in the tumor substance. The medial aspect of the mass was firmly incorporated in the pedicle of the right kidney. The mass with the kidney and ureter was removed. A great deal of bleeding was encountered, and the patient was given 1500 cc. of whole blood in a short time while on the table. The patient's condition was so critical that it was felt inadvisable to continue further. Eight large Kelly clamps were left applied to the bleeding points and the abdomen closed in layers below the handles of these clamps. Heavy dressing were applied. The patient left the operating table in a critical condition and expired two days later. Permission for an autopsy was not granted.

PATHOLOGIC DATA

Gross: The specimen consisted of a lobulated mass of yellowish tissue which measured 20 x 16 x 12 cm. The kidney was incorporated within this mass. Most of the lobulations were fairly well encapsulated and on repeated sections showed fat only. Others were more solid and glistening. Some were discolored. The kidney was not involved.

Microscopic examination: The sections showed curious combinations of adult and fetal cells which were intimately related to a quanity of elongated spindled cells. Many of these cells showed no definite cytoplasm, and the nuclei were difficult to identify. In several areas there were condensations of hyperchromatic nuclei which resembled giant cells. Some myxomatous tissue was present. On the whole this was more cellular than the sections of July 31, 1942.

Case Report II: The patient, a 60 year old, white executive, was admitted to Mercy Hospital, Pittsburgh, Pennsylvania, for the first time on May 10, 1937. At that time his chief complaint was an ulcer on the left lower leg.

In August 1935 he had first noticed a small discolored area within the skin of the calf of the left leg. This was ignored until October 1936, at which time an associated soreness developed. The dermatologist whom he consulted excised a small mass underlying the discolored area. The pathologic report by Dr. H. H. Permar diagnosed liposarcoma. This was confirmed by Dr. James Ewing when sections were sent to him in New York.

A second mass was excised by the dermatologist in March of 1937. Following a second report of liposarcoma, irradiation therapy was instituted, and the patient finally consented to hospitalization on May 10, 1937. The remainder of the history disclosed little of interest, other than a thyroidectomy in 1932.

A physical examination of this patient revealed a well developed, well nourished, white male in no visible distress. No abnormalities were noted with the exception of a large indolent ulcer with erythematous edges which was present upon the skin of the calf. Immediately adjacent to this were two small, well healed incisions. A small nodule was present under the skin below the knee and another was palpable in the tendons of the hamstrings. The inguinal lymph nodes were moderately enlarged.

Laboratory studies showed an erythrocyte count of 4,700,000 and a leucocyte count of 7,800 with 67 per cent polymorphonuclears, 20 per cent lymphocytes, 10 per cent monocytes, and 3 per cent eosinophiles. The urinalysis was normal. Blood chemistries were: non-protein nitrogen 30.4 mgm. per cent; blood sugar 92 mgm. per cent; blood calcium 9.4 mgm. per cent and blood phosphorus 3.9 mgm. per cent. The Wassermann and Kahn tests were negative.

On May 12, the leg was amputated at the junction of the middle and lower thirds of the femur. At the same time a thorough resection of the inguinal lymph nodes was undertaken.

The pathologic diagnosis was (1) Liposarcoma of left leg with metastasis within the leg, and (2) Chronic inguinal lymphadenitis. It was assumed that the metastasis within the leg was by way of the lymphatics, in spite of the lack of metastatic lesions within the inguinal lymph nodes.

Post-operatively the convalescence was uneventful, the stump healing by first intention. The patient was discharged on June 22, 1937.

Upon four occassions following this he was admitted to the hospital for excision of nodules developing within the stump or within the scar. At operation in all four instances the masses were found to be firm and well encapsulated. In all four instances a wide excision was done so as to include much of the surrounding, apparently normal, tissue. Each time the pathologic diagnosis was liposarcoma. The wound always healed by first intention.

The sixth admission was on July 1, 1941. At this time a tender, egg-sized mass was observed at the lateral aspect of the stump. It was also noted that the patient had lost twenty-eight pounds in weight, the first evidence of other than local disease.

On July 7, 1941 the left femur was disarticulated at the hip joint. Again the post-operative course was easy with not so much as an elevation of temperature. The pathologic report was liposarcoma, with a more desmoplastic aspect than before. The patient was discharged on August 6, 1941.

The seventh admission occurred on December 14, 1941 when multiple nodules were discovered within the scar. These were once again excised with much of the surrounding tissue and radium was implanted locally. The pathologic diagnosis was unchanged.

The eight and final admission was on May 13, 1942. An examination revealed a large mass extending from the stump up over the medial aspect of the thigh, along the left side of the symphisis, and extending into the femoral region.

Under nitrous oxide, oxygen and ether anesthesia an interinnomino resection was done. Three hours post-operatively the patient suddenly gasped and died while being visited by the attending surgeon. Permission for autopsy was not granted.

PATHOLOGIC DATA

A total of ten specimens were sent for examination from January 4, 1937 to May 13, 1942. This included the original lesion under the skin of the calf to the inter-innomino resection.

Gross: In general the tumor masses were nodular, yellowish, and moderately firm. They were fairly well demarcated, and ranged in size from 1 to 8 cm. in diameter. Occasionally a rich plexus of veins was seen about their borders. As time went on these tumors increased in density and paleness, although even in later instances some appeared yellow and fatty.

Microscopic examinations: The microscopic aspects were consistent in that

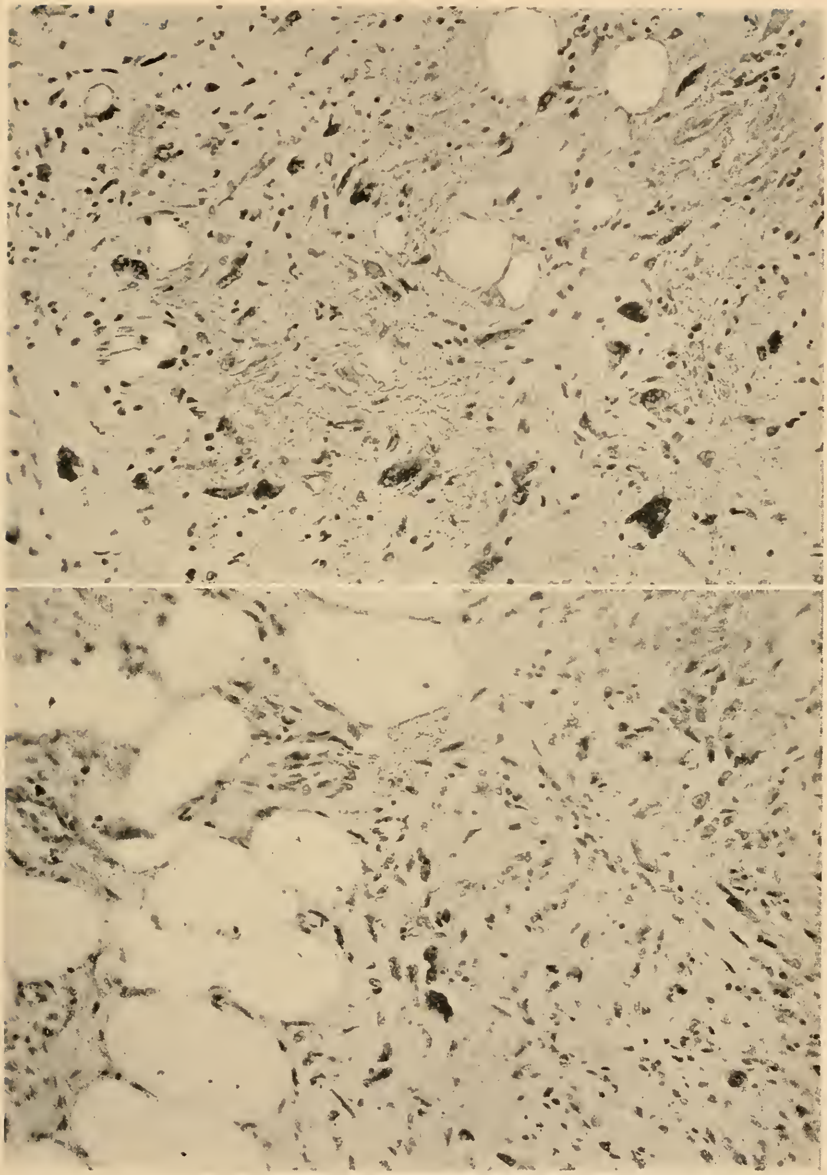

FIG. 1. *a* (top) Case 1, *b* (bottom) Case 2.

in all sections the origin of the tumor could be made out as adipose tissue. The cells were large, from oval to spindle forms, with varying amounts of cytoplasm. At first there was more of a tendency toward loose arrangement, but later dense masses or whorls were more prominent. The nuclei were consistently large and hyperchromatic. Mitotic forms were numerous in all sections.

CONCLUSIONS

Liposarcomas occur with equal frequency in either sex, and in any age group. The predominant sites of occurrence are in the soft tissues of the legs and the retroperitoneal space. There is slow growth for many years with a sudden acceleration in their rate of development, and a great tendency of recurrence after excision. The encapsulation and ready removability do not at times suggest their malignancy. It is of utmost importance to keep in mind that their histologic structures are variable. Its response to roentgen therapy is much like that of other sarcomas.

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A CASE OF SICKLE CELL ANEMIA COMPLICATING PREGNANCY*†

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Sickle cell anemia, also known as sicklemia, meniscocytosis, drepanocythemia, or Herrick's syndrome, is a true hemolytic, hereditary anemia, transmitted as a Mendelian dominant. It is characterized by the presence of bizarre, crescent-shaped or sickle-shaped red corpuscles. The clinical symptoms are said to result entirely from the peculiar deformity of the red corpuscles which are destroyed in excessive numbers.

This disease was first described as a clinical entity by Herrick in 1910. Huck demonstrated that the sickling phenomenon is an inherent trait of the red cells rather than one of the plasma, while Hahn and Gillespie showed that the sickling could be produced more readily when the red cells were removed from contact with oxygen. There is apparently no relationship between the sickling trait and the various blood groups; and the sickling occurs irrespective of the medium in which the cells are placed.

The disease is confined almost entirely to the negro race, though a few reports have cast some doubt as to whether or not it can occur in other races. Killingsworth and Wallace (2) analyzed the studies performed in ten states and found that of 9,658 negroes examined, 684 or approximately 7 per cent exhibited the sickle cell trait, but they point out that only one out of 15 of these develops sickle cell anemia (.45 per cent). The sickling trait has been reported at all ages (average age 13 years), and it apparently predominates in females. Young adults are more likely to develop the anemia after puberty, and the older the person, the less likely is he to develop the disease. Kracke (4) states that as a patient with sickle cell anemia grows older, the attacks become lighter, both in frequency and severity.

Davison quotes the following figures regarding frequency of occurrence of the various signs and symptoms among a group of 60 patients during an acute exacerbation:

Abdominal and splenic pain—55%—may simulate appendicitis or a ruptured viscus
Joint pains—51%
Fever—33%—may suggest typhoid fever or tuberculosis
Apical cardiac murmur—75%
} may suggest rhuemátic fever, subacute bacterial endocarditis, or septicemia

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* From the Baltimore City Hospitals.

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Cardiac enlargement—48%
Pulmonary congestion—39%
Enlarged tonsils and cervical and axillary lymph nodes—55%
Chronic leg ulcers—39%—frequent in adults—may imitate syphilis
Greenish sclerae—50%
Retarded sexual development—17%

For the most part, the anemic individuals give a history of recurrent attacks of weakness, fatigue, or circulatory symptoms suggestive of anemia. Upper abdominal pain is thought to result from recurrent infarctions of the spleen.

Up to the present time, only 24 cases of pregnancy in women with sickle cell anemia have been reported in the literature. Since this is apparently a relatively rare condition, the following case is presented:

CASE REPORT

E. W., a 23 year old colored, para 3–1–0–2, housewife, was admitted to Baltimore City Hospitals on August 15, 1947, in approximately the thirty-sixth week of pregnancy. She had a temperature of 100.8 F., a blood pressure of 168 systolic over 102 diastolic. The patient complained of weakness and generalized aching, which had lasted one week. The family history was non-contributory, other than revealing that the patient was an only child. The past history revealed that she had had icteric sclerae for a short period of time two years previously and that during the past two years, she had had recurrent aches in the hips, thighs and knees, unaccompanied by fever or swelling.

The patient's menstrual periods began at the age of 14, and were described as being normal. Past obstetric history revealed three previous full term deliveries in 1941, 1942 and 1945; the child born in 1941 subsequently died of pneumonia. In 1944, the patient was delivered of a 4 pound baby, which died thereafter of colitis. The patient experienced slight dyspnea with the previous pregnancies; and during the last month of the pregnancies carried to term, she noted some swelling of the ankles.

During this pregnancy she had noted dyspnea, and, for three weeks prior to admission, she experienced recurrent lower abdominal cramps. One week before admission, she noted weakness, generalized aches, and spots before the eyes. There was no history of recent upper respiratory infection, dysuria, chills, fever, nausea or vomiting.

A physical examination on admission revealed a temperature of 100.8 F., a pulse of 104, a respiratory rate of 28 and a blood pressure of 168 systolic over 102 diastolic. The patient, a well developed, slightly obese colored female, was in no acute distress. The palms of the hands, the soles of the feet, and the mucous membranes were very pale. The sclerae of the eyes had a yellowish tint; the retina of the left eye showed pigmentary degenerative changes. The heart was enlarged and to the left. There was a short, blowing, non-transmitted, systolic murmur at the pulmonic and apical regions; the aortic second sound was reduplicated. The rhythm was regular. An examination of the abdomen was negative except for pregnancy findings. Edema was not present, and there were no leg ulcers.

The patient was placed on penicillin therapy and put on an intensive, pre-eclamptic regime. Studies of the blood revealed the following: the serologic test for syphilis was negative, the Hematocrit, 16; the Hemoglobin, 5.0; the red blood count, 1.53, the color index, 1.01, the mean corpuscular volume, 104; the mean corpuscular hemoglobin content, 33; the white blood count, 6,200; the differential: Segmented, 75; eosinophiles, 1; lymphocytes, 20; monocytes, 4. The smear revealed anisocytosis and poikilocytosis, occasional erythroblasts and numerous normoblasts. There was 7–8 per cent reticulocytes. The sickle cell preparation revealed 7–8 per cent sickling in twenty minutes and about 70 per cent in sixteen hours. Bleeding and clotting times were normal. The icteric index was 7.5. The urine was positive for urobilinogen and showed a 1 plus albumin. Both the patient and her husband were found to be Rh positive.

On her fifth hospital day, after the fetal heart had not been heard for several hours, the patient commenced labor and after a total labor of one hour and forty-eight minutes delivered spontaneously from left occiput anterior, a stillborn female child weighing 5 pounds 5 ounces. The baby was born with four complete loops of umbilical cord tightly applied about its neck. Both the baby and the placenta were sent to the pathologic laboratory for examination. The baby showed compression atelectasis of both lungs, ascites, and hydrothorax. The erythrocytes within the blood vessels of the chorionic villi were round and showed no sickling tendency, while the erythrocytes within the blood vessels of the decidua showed definite sickling tendency. After delivery, the patient's spleen was palpable 3–4 fingerbreadths below the left costal margin. The patient was given 1500 cc. of citrated whole blood and placed on liver and iron therapy. Roentgenologic studies revealed cardiac enlargement. The long bones showed minimal changes suggestive of sickle cell anemia.

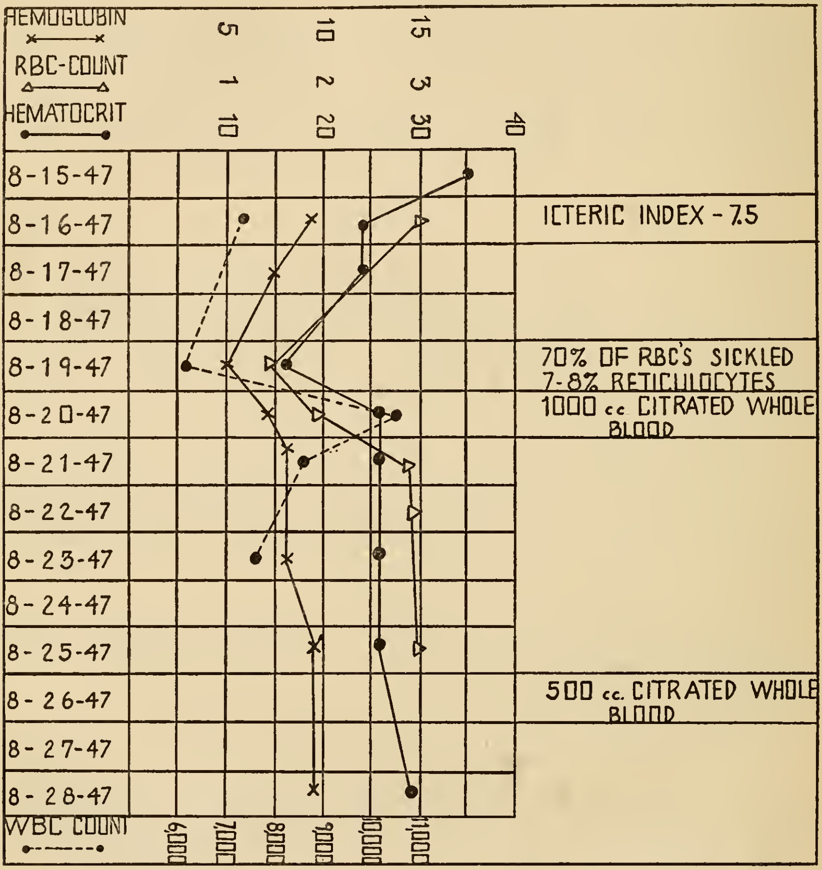

FIG .1. The various blood changes which occurred in the reported case

The patient was discharged on her fifteenth hospital day at which time her temperature had returned to normal. Her blood pressure had stabilized at 130 systolic over 90 diastolic, and her hematocrit had risen to 29.

Recently Hodges and Bernstine (1) reported 4 cases of pregnancy in women with sickle cell anemia. Two of these cases had signs and symptoms of central nervous system involvement occurring immediately after delivery and presenting aspects resembling toxemia of pregnancy. They critically reviewed all the cases of sickle cell anemia in the literature to that date, except the first two cases reported in 1924 by Sydenstricker (13) who did not furnish clinical descriptions. They found the following facts:

(1) All but 3 of the patients were less than 30 years of age; the oldest was 35 and the youngest was 16 years.
(2) Six died as a result of a complication of pregnancy.
(3) The clinical course of 10 cases suggested thrombotic phenomena with 7 having signs and symptoms of pulmonary infarction; 2 of cerebral thrombosis; and 3 of thrombi in the legs or pelvic vessels and abdominal viscera.
(4) Nine gave histories of spontaneous abortions, miscarriages, or both.
(5) Thirty-three pregnancies were reported as having progressed beyond 7 calendar months with 24 infants surviving and 9 dying either in utero, at delivery or soon afterward. Twenty of these infants were examined for sickling and 14 were reported as positive.
(6) The blood pressure was reported in 13 cases. Of these 7 had hypertension (diastolic blood pressure over 90mm of mercury).

In regard to the above discussion, this patient was 23 years old, suffered no thrombotic phenomena, and was discharged from the hospital in fairly good condition. She has had five pregnancies which have progressed beyond 7 calendar months and at present has two living children. The baby delivered at the Baltimore City Hospitals did not have sickle cell anemia. The patient's blood pressure had been above 130mm systolic pressure and 90mm diastolic pressure during the majority of her hospital stay.

SUMMARY

(1) One case report of sickle cell anemia in preganancy is added to the 24 published cases.

(2) This patient presented signs and symptoms of toxemia of pregnancy.

(3) This case represents a crisis occurring during pregnancy with at least a temporary response to transfusion therapy.

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VITAMIN K AND LATE TONSILLAR HEMORRHAGE* †

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Recently several reports have appeared in the literature concerning the value of vitamin K in reducing the incidence of late tonsillar bleeding. The advocates of this therapy assume that late tonsillar bleeding is the result of a salicylate-induced hypoprothrombinemia, which the administration of vitamin K would prevent.

The authors at first administered vitamin K along with the usual Aspergum therapy in an attempt to reduce the incidence of late tonsillar bleeding among their patients. Since an early analysis failed to substantiate the value of vitamin K in this regard, a controlled series was undertaken to study the problem.

Although it has been almost eighteen years since Dam (3) began his classic experiments leading to the discovery of vitamin K, the clinical uses of this vitamin have been relatively slow of acceptance. Reliable clinical and laboratory experiments have proved that salicylic acid or salicylates will prolong the prothrombin time when administered in large doses to animals or man. This occurs whether a normal or abnormal prothrombin time is originally present (5, 11, 4, 7). Clausen and Jager (2) state that salicylates block the utilization of vitamin K and that this is probably the basis of the hypoprothrombinemic effect. Several groups of investigators, working independently, concluded that vitamin K would prevent a salicylate-induced hypoprothrombinemia (5, 11, 7, 12). Shapiro, et al. (13), found that 1 mgm. of vitamin K furnishes the most reliable protection to 1 gm. of salicylate. According to Clausen and Jager (2), vitamin K has no effect on hypoprothrombinemia previously established by salicylate therapy, but this is not accepted by all investigators. Butt, et al. (1), showed that when sodium salicylate was given in doses of 10 gm. a day for uncomplicated rheumatic fever cases, the prothrombin was not reduced to a hemorrhagic level.

Quick (9) reports a patient who suffered a marked depletion of prothrombin from small doses of salicylates. He feels, however, that in such cases another dysfunction is present. He further states that "in the normal patient vitamin K will restore and maintain the normal prothrombin level, but in such patients the level without vitamin K is not low enough to cause hemorrhage." Vitamin K, given in relatively large doses, causes no demonstrable hyperprothrombinemia in rabbits, dogs or man, even if the individual

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is suffering from a congenital hypoprothrombinemia. If the prothrombin were increased above normal, it would not necessarily affect the coagulation time, since the trigger substance in the clotting mechanism is thromboplastin. Quick (9) emphasizes that hypoprothrombinemia becomes clinically significant only if the prothrombin falls below 20 per cent of normal, as it is not until then that the patient is in danger of hemorrhage. However, if the vascular response remains normal and no trauma occurs, abnormal bleeding is not likely to occur even then.

RELATIONSHIP TO TONSILLECTOMY

In adenotonsillectomy, a sizable area is left raw and uncovered. This soon becomes infected superficially by the invasion of the normal bacterial flora of the mouth and throat. The reaction to the bacterial invasion, plus the normal hemorrhagic and serous exudation, produces a crust which finally covers the entire raw surface. This crust is continually under stress and strain by the normal movements of the regional muscles. Within twenty-four to forty-eight hours, granulation tissue begins to form beneath the crust. This tissue grows from below upward, a process requiring from five to eight days. Displacement of the crust results. As the crust slowly disintegrates and becomes dislodged, the granulation tissue is exposed. Since the granulation tissue is highly vascular, trauma, whether by food bolus or by movements of the tongue or regional muscles, may produce hemorrhage. As mucous membrane slowly proliferates to cover this granulating surface, the possibility of bleeding from the area is reduced accordingly.

Singer (14), who formerly practiced in Austria, where salicylates are not used in the postoperative management of tonsillectomy cases, made the observation that late tonsillar hemorrhage was much less common in the clinics of Central Europe than it was in this country. The only significant difference in the management of the cases on the two continents, according to him, is the use of aspirin here. In Central Europe aminopyrine is the analgesic drug in vogue.

Neivert (3) studied a series of 283 patients undergoing tonsillectomy. He administered both vitamin K and acetylsalicylic acid throughout the postoperative period, and found on examination five to seven days postoperatively, that the incidence of late tonsillar hemorrhage was 1.4 per cent. Previous to this, his other patients had received abundant salicylates alone, and his incidence of late tonsillar hemorrhage was almost 10 per cent. He concluded that salicylates must be avoided or must be administered simultaneously with vitamin K.

In 1946, Preston (8) in his series reported that secondary tonsillar hemorrhage occurred in from 4.29 to 7.07 per cent of the cases when he used aspirin alone over a five-year period. By the simultaneous administration of vitamin K, he obtained 2.08 per cent late bleeding among 385 patients.

Neither of these workers described the exact method employed to determine the percentage of late tonsillar bleeding, other than their observations approximately one week postoperatively. This point of possible error is stressed since, in the series here reported detailed questionnaires were used with careful and persistent follow-up by mail and phone. The percentage finally determined showed marked discrepancies with the original figures obtained from case records without the questionnaire.

It was difficult to believe that the relatively small doses of salicylates given during the usual postoperative course of adenotonsillectomy could deplete the prothrombin to such an extent as to place otherwise normal patients in danger of hemorrhage. In addition, as pointed out by Quick, (10) the bulk of the evidence put forth to prove that aspirin does reduce the prothrombin is based on the prothrombin time of diluted plasma, which, even in the hands of skilled technicians, is inaccurate.

The need for additional accurate clinical and laboratory evidence was realized. It was not practical to determine the prothrombin time routinely on adenotonsillectomy admissions, or at the time of the postoperative visits to the office or clinic, especially at the time of any late bleeding. Because of this, a series was begun to test the protective value of vitamin K in relation to salicylate therapy and secondary postoperative hemorrhage in adenotonsillectomy on a clinical basis.

THE SERIES

A total of 687 adenotonsillectomy patients was studied.* The usual pre- and postoperative examinations and observations were recorded on each. In addition, a questionnaire was mailed to each patient (or the parent in the case of children) approximately one month postoperatively. Self-addressed stamped envelopes were enclosed to facilitate return of the questionnaire, and repeated telephone follow-up inquiries were made until the questionnaire was returned.

By definition, bleeding which occurred within twenty-four hours of the time of operation is considered primary bleeding, and results either from inadequate hemostasis at the time of operation or from the loss of free ligatures at bleeding points. Bleeding which occurs thereafter, and especially after forty-eight hours, is considered late or secondary bleeding. This series deals only with the late (secondary) type of bleeding. All reports of bleeding were confirmed, but recorded in this series, no matter how minimal.

Group 1 included 216 patients who used Aspergum as desired throughout the postoperative period. No other medication was prescribed or used, except codeine in adults (as it has been shown that codeine does not affect the prothrombin time). Of this number, 21 patients reported secondary (late) bleeding. This represents an incidence of 9.7 per cent (see Fig. 1).

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* Previously the authors published the results obtained in 504 cases of adenotonsillectomy (4).

In Group II, 128 patients took 10 mgm. of Synkayvite* (Synthetic Vitamin K) twice daily for seven days postoperatively along with Aspergum as desired. Codeine was prescribed for the adult patients. In this group, 13 patients reported postoperative bleeding. This represents a 10.1 per cent incidence of late tonsillar bleeding.

Group III consisted of 343 patients who were not permitted to use Aspergum at all. No Synkayvite was taken by them. Codeine was prescribed for the adults in the first 184 cases of this group but no other medication was given. Of this number, two cases of secondary postoperative bleeding were reported. Of the remaining 159 patients in Group III, those over 12 years of age were advised to use Tyrozets** postoperatively for discomfort, instead of codeine. In these latter cases no postoperative bleeding was reported and the fossae were uniformly cleaner and free from excessive granulation tissue. In one of the two patients in Group III with late tonsillar bleeding, the prothrombin time was found to be normal at the time of the bleeding. Group III showed an incidence of 0.58 per cent of late tonsillar bleeding.

	GROUP I	GROUP II	GROUP III
Total cases	216	128	343
Number exhibiting late bleeding	21	13	2
Percentage of late bleeding	9.7	10.1	0.58

FIG. 1. Analysis of Cases

Further analysis of the series showed that the average voluntary consumption of Aspergum during the postoperative period was 16 tablets (each containing 3½ gr. of aspirin), or a total of 56 gr. of aspirin. This figure is representative of both those who bled and those who did not. The maximum amount of Aspergum taken by any one patient in the series was 112 tablets, or 392 gr. of aspirin. Ironically, this patient did not bleed.

Approximately one-half of those that bled required medical attention (16 out of 34). Only 3 of these required general anesthesia and hospitalization to effect control of the bleeding.

In this series late tonsillar bleeding occurred most often during July, August and September, but no adequate scientific explanation can be offered for this. Several factors have been suggested to account for the increased incidence during the hot season. Secondary infection and putrefaction may be more marked in hot weather. Dehydration is also more common, as the water loss is greater and the intake is usually restricted by the patient as a result of local discomfort on swallowing.

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* Hoffman-LaRoche, Inc.

** Tyrozets (Sharp & Dohme) are troches containing benzocain and tyrothricin.

The majority of the cases in each group of the series were operated upon during the warm months so that equivalent climatic conditions prevailed. All patients were operated upon by one or the other of the authors by an identical technique which consists of sharp dissection throughout and the control of all bleeding points by No. 0 or No. 1 plain catgut free ligatures.

SUMMARY AND CONCLUSIONS

A comparison of Groups I and II in this series fails to reveal any protective action afforded by vitamin K (Synkayvite) in late tonsillar hemorrhage.

By discontinuing the use of Aspergum postoperatively, it was possible to reduce the incidence of late tonsillar hemorrhage from 9.7 per cent in Group I to 0.58 per cent in Group III.

It is generally agreed that the simultaneous administration of vitamin K will protect a normal individual against a salicylate-induced hypoprothrombinemia; but the authors believe that in the vast majority of cases hypoprothrombinemia is not the cause of late tonsillar hemorrhage.

It is felt that the high incidence of late tonsillar bleeding in patients using Aspergum is probably caused by some local effect on the wound.

One hundred and fifty-nine patients of Group III were studied to determine the benefit of an antibiotic (tyrothricin) locally on the healing of tonsillectomy wounds. It is believed by the authors that a cleaner fossa with a minimum of granulating surface results, thus lessening late tonsillar bleeding. In this sub-group there were no cases of postoperative bleeding, and it is therefore felt that secondary infection plays a major role in the causation of late hemorrhage.

This study has proved conclusively to the authors that analysis of a series of such patients absolutely requires a careful canvass by specific questionnaire and persistent follow-up. By this means it was determined that many patients failed to follow postoperative instructions, including prescribed medications, even when presented upon discharge from the hospital with a printed list of detailed instructions containing specific prescriptions. Therefore conclusions drawn from office and clinic records alone showed gross discrepancies.

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THE RELIABILITY OF PERCUSSION OF THE LEFT HEART BORDER*†

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There are three methods of determining the left cardiac border. They are: (1) palpation of the apex beat; (2) percussion of the border; (3) roentgenologic examination.

No one of these methods should be depended upon exclusively, since each of them has its disadvantages. Only a combination of them will lead to an accurate determination of the left cardiac border, which is changed in so many pathologic conditions.

In normal individuals, the apex beat lies just within the border. However, it will not be palpable when the walls of the chest are thick, the lung emphysematous, or the heart not beating vigorously. Also in such pathologic conditions as pericardial effusion, the apex beat is not palpable.

Percussion of the cardiac outline has been the cause of much controversy. Many physicians prefer to rely on roentgenography and the position of the apical impulse. Unfortunately, in many instances no cardiac impulse is palpable, and often the employment of the roentgen-ray is impossible. A roentgenologic laboratory may be miles away; but the physician has his hands and ears with him at the bedside of the patient.

The area of cardiac dullness does not conform accurately in size or shape with the cardiac outline as determined by roentgenography. However, there is a rough approximation of the findings which these two methods give of the size of the heart (1).

In the observations to be reported in this paper, an attempt was made to find the outermost point of the left heart border (relative dullness) by means of percussion, and then to compare this result with the findings of the roentgenographic method. Only lean male patients were used as subjects. The majority of them had hearts within normal limits as to size. Some had hypertrophied hearts, and three had rather small hearts.

The conventional, moderately forceful percussion was carried out in the fourth and fifth interspaces, in the direction from the left anterior axillary line towards the midline. The pleximeter finger was held parallel to the expected border. Moderate percussion was used for the left border; and especial care was exercised to deliver the percussion blow in a vertical direction. Unless the percussion is performed with great care, the left border

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will be overestimated (3). The outermost point of the left cardiac border was determined. From this point, a sagittal line was drawn towards the heart, and a marker was attached to the skin at the distance of 4 cm. This was done to insure the visibility of the marker on the roentgenograph.

Roentgenographs were then taken at a distance of six feet, and the position of the marker was compared with the outermost point of the left cardiac silhouette.

The results were as follows: the diameter of the chest and the margin of error were recorded in each case. The plus sign in the column of deviation means that the percussion result exceeded the border as found roentgenographically. The minus sign indicates that the percussion point was inside the roentgenographic border.

FILE NO.	ANTERIOR-POSTERIOR THORAX DIAMETER	DEVIATION
919	25 cm.	+4 mm.
920	23 cm.	−13 mm.
921	24 cm.	+6 mm.
923	24 cm.	−16 mm.
930	26 cm.	+16 mm.
935	27 cm.	−4 mm.
936	21 cm.	−2 mm.
938	24 cm.	−12 mm.
944	23 cm.	−18 mm.
945	23 cm.	−12 mm.
948	24 cm.	+3 mm.
956	24 cm.	0
957	20 cm.	+15 mm.
966	22 cm.	+1 mm.
971	23 cm.	+15 mm.
979	26 cm.	0
991	24 cm.	+10 mm.
1000	22 cm.	+9 mm.
1007	25 cm.	+31 mm.
1009	25 cm.	−8 mm.
1012	21 cm.	−8 mm.
1017	20 cm.	−7 mm.
1020	24 cm.	+16 mm.
1021	23 cm.	−13 mm.
1024	22 cm.	+9 mm.

From the number of cases examined (25), one could deduce the following: (1) the heart size was overestimated in about as many cases as it was underestimated; (2) the margin of error was not more than 1 cm. in 56 per cent of the cases and not more than 1.5 cm. in 80 per cent of the cases; (3) the greatest error from the percussion result occurred in individuals who showed a very large anterior-posterior diameter of the chest.

CONCLUSIONS

From these observations, the authors conclude that the percussion of the left cardiac border should not be discarded as unreliable, but should be used in conjunction with other methods of determination.

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OBSTETRIC CASE REPORT*†

The patient, a 34 year old, para 2–1–0–2, was first seen early in her pregnancy and received the usual prenatal care. Except as noted below, the pregnancy had been uneventful until the day of admission. The patient's past obstetric history was pertinent in that her first pregnancy was terminated at 34 weeks because of placenta previa. Following this, she had two uneventful full-term pregnancies, giving birth to living infants.

When the patient was approximately 37 weeks pregnant, abdominal palpation revealed that she had a "transverse lie," right acromio anterior. At this time no attempt was made to do an external version. It seemed probable that the baby would revert to the original position, so no good would be accomplished. Also there was the real danger of grave injury to the uterus by the necessary manipulations. Because of the presentation and because of the obstetric history, a roentgenograph was requested, to determine the approximate size of the baby, the presence or absence of fetal abnormality or demonstrable tumors obstructing the birth canal and, finally, the location of the placenta. The roentgenologist confirmed the diagnosis of transverse lie, found nothing abnormal about the baby or the pelvis, and reported that the placenta was on the lateral and anterior aspect of the uterus and probably under the site of the original incision in the uterus.

Three days later, before the patient had been seen again, her husband reported by telephone that she was complaining of a severe constant pain in the epigastric region. He was advised to bring her to the hospital immediately, and she was admitted about three quarters of an hour later, or about one and three quarter hours after the onset of pain. Her general condition at this time was good, pulse, temperature, and respirations being within normal ranges and the blood pressure 122 systolic over 74 diastolic. The abdomen was distended; there was rigidity of the abdominal muscles, and the entire abdomen was tender. There was no bleeding from the vagina. Some shifting dullness was present in the flanks.

QUESTION: (a). What is the most probable diagnosis?
(b). What treatment should be carried out?

DISCUSSION

From the information given above, it is quite apparent that there had occurred a sudden "accident of pregnancy." The two most probable diagnoses would be *abruptio placenta* and *rupture of the uterus*. The previous obstetric history, the presentation of the baby, the location of the placenta and the presence of free fluid in the abdomen (shifting dullness) point so strongly to the latter of these two, that no further evidence is needed. When the uterus has once been incised, there is always the possibility that it may rupture in a subsequent pregnancy or labor, and this possibility increases almost with geometrical progression with succeeding pregnancies. There is some evidence that this danger is increased when the placenta is under the scar in the uterus.

The recommended treatment in this case is an elective cesarean section near term, to be accompanied by sterilization. This was the course planned;

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but because of the acute accident, it could not be carried out. In the case as presented then, the proper treatment would be to prepare to treat shock by having blood available, and to open the abdomen as soon as arrangements could be made. Subsequent procedures would depend upon the findings after a laparatomy was done.

ACTUAL TREATMENT

The actual treatment was as outlined above. When the abdomen was opened, a large rent was found in the uterus. The entire product of conception was free and intact in the abdominal cavity. The membranes were ruptured artificially, and a dead child removed. After this, the placenta and membranes were lifted out with no difficulty. Upon inspection of the uterus, the rupture was found to be quite extensive, and a hysterectomy was decided upon. This was done without too much difficulty; and the patient, who had received three pints of blood during the operation, left the table in good condition. The recovery was uneventful.

THE EXTRAMURAL POSTGRADUATE TEACHING PROGRAM OF THE UNIVERSITY OF MARYLAND SCHOOL OF MEDICINE*

MILTON S. SACKS, M.D., F.A.C.P.†

BALTIMORE, MARYLAND

The School of Medicine University of Maryland has embarked on a program of postgraduate medical education which, it is believed, will become of greater importance with the passage of time. Postgraduate medical education, always recognized as important, was undoubtedly spurred on by the needs of returning medical veterans of the last war. It was, of course, obvious that numerous and diverse approaches to the problem existed. However, above and beyond the extraordinary needs of a particular time, was the basic and continued necessity for postgraduate medical education. This had always existed for those physicians whose locale of practice did not enable them to maintain close contact with teaching centers. The Committee on Post Graduate Studies of the Medical School was aware of the difficulties which faced physicians practicing in rural areas and prevented their absenting themselves from their duties. In an effort to circumvent this situation, the Committee decided to explore the possibility of establishing postgraduate seminars in centrally located areas throughout all of the counties of the State. In order to assess the value and need for such a program, questionnaires were sent to the secretaries of all county medical societies in June, 1947. As a result of the interest shown by various county societies the present program of extramural courses has been developed.

The first series of postgraduate seminars was begun on October 16, 1947 under the auspices of the Prince George's County Medical Society, at the Prince George's General Hospital, Cheverly, Maryland. A group of thirty-seven physicians, members of this county's and neighboring county medical societies are at present pursuing postgraduate studies. A similar group, composed of forty members of the Washington County and adjacent county Medical Societies, has been meeting monthly since December, 1947 at the Washington County General Hospital in Hagerstown. The third group, representing an amalgamation of Howard and Carroll County physicians, comprises a total of twenty-one physicians who have been meeting monthly since December 1947, at the University Hospital. Thus, during the first experimental year of this program, ninety-eight physicians, representing approximately one-seventh of all the practitioners in the various counties

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* Received for publication February 2, 1948.

† Secretary, Post Graduate Committee.

of Maryland, are participating in a voluntary program for continued education. This cannot but have a salutary effect upon the health and well-being of the citizens of the State.

It may be helpful to review briefly the procedure followed in these programs: The sessions are planned on a monthly basis. It was hoped to begin them in October and continue through May, making a total of eight sessions for the fall, winter and spring seasons. Because of unavoidable delays in planning, several courses of the current year did not start until December. A more uniform beginning may be expected for succeeding seasons. It has been the policy of the Committee to invite the members of the various county societies to indicate their preferences as to the day of the meeting, its location, and the subjects desired. The latter have usually been selected after a poll of the individual members of each society. This information in turn is submitted to the Post Graduate Committee which then undertakes to prepare the schedules. In each instance, depending upon the topic selected, a chairman for the particular session is chosen on nomination by the head of that Department of the Medical School which covers the material to be discussed. In all instances, the chairman is an individual whose professional accomplishments permit him to deal authoritatively with the subject. The chairman of the panel in turn selects as many men as he may deem necessary to present a well-rounded review of the subject. Technical adjuncts, such as roentgenographs, lantern slides, pathologic specimens and motion pictures are used as the occasion demands. All meetings begin at 3:30 p.m. and run for 2½ hours. During this time a general review of the subject is presented, with emphasis upon recent developments. Following a plan originally established with the Prince George's Medical Society, it has become customary to adjourn from six to seven p.m. for dinner at the institution in which the seminar is being given. This has constituted a pleasant interlude, permitting closer professional and social contacts. From seven until nine p.m., an informal round table, question-and-answer period is held.

During the 1947–1948 season, the following topics are being presented, each occupying one whole session: Orthopedics, Obstetrics, Cardiology, Psychosomatic Medicine, Pediatrics, Gynecology, Diabetes, The Acute Surgical Abdomen, Allergy, Hypertension. The response, both on the part of the physicians taking the courses and of those giving them, has been quite enthusiastic. For the current session, an individual registration and tuition fee of $15.00 has been charged for the entire series.

The Committee hopes to be able to expand this program during the 1948–1949 season. Preliminary contacts with the officers of county medical societies will be made in the early Spring of 1948 in order to permit members of the societies to consider possible participation in next year's program. Experience has indicated that a group of at least twenty-five physicians must constitute the minimum number for whom panels can be arranged. In those instances where county medical societies have a membership which falls beneath this minimum, group affiliation with neighboring societies is advisable.

The plan of organization permits of great elasticity in the selection of topics. There need be no repetition of subjects in any one area from one season to another. Beginning very shortly the Committee plans to record the proceedings of each session and to publish them, in some form for distribution to physicians taking the course.

The continuation and success of the entire program depend largely upon the extent to which it fulfills a definite need in the improvement of the overall medical care of the citizens of Maryland.

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* The names listed above are officers for the term beginning July 1, 1947 and ending June 30, 1948.

A REPORT ON THE MEDICAL LIBRARY RARE BOOK RESTORATION FUND

The response to the appeal for alumni contributions to restore the bindings of the library's historical collection has been generous beyond all anticipation. To date, an amount totaling $4175.00 in cash and pledges has been received ($1651 in cash; $2524 in pledges). The School of Medicine can rightfully feel proud of its alumni and friends for their tangible interest in this project.

Contributions are still coming in. In the February 1948 issue of MARYLAND, a preliminary list of names was published, showing contributors to December 1947. That list has been enlarged with names added since (through the middle of February 1948) which appear herewith. Later additions will appear in the Bulletin.

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I pledge an annual contribution of ...... for five years for the restoration of the rare books of the Medical Library. Enclosed is my 1947 contribution.

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JOURNALS WANTED

Members of the library staff wonder whether library patrons are aware of the serious problem created by the loss of volumes or single issues of medical journals. In prewar days, when a current volume was lost, the librarian collected its price from the offender (if he were known) and ordered a replacement copy of the journal. Today that is impossible in most instances. Because of publishing difficulties, subscriptions are strictly limited; and extra copies of unbound issues or complete volumes of journals are not procurable. (Of course this is invariably true of older volumes which are out of print.) Thus the loss of any copy of a current journal today means that it will probably *never* be available to the library. Such losses, multiplied, produce serious breaks in the periodical files. Yet these files should be complete in any good medical library.

We make this explanation for two reasons: to appeal for conscientious attention to the safe return of library material; and to ask from alumni contributions of medical journals which they do not personally wish to keep. Such contributions may serve both to supply missing numbers of periodicals and to provide duplicates useful in the increased demands made on the library. Nothing goes to waste; any duplicates in excess of our own library's needs are used, through the Medical Library Association, for exchange with other medical libraries, American and foreign. We shall be most appreciative of any journals supplied by interested alumni.

The following is a list of specific issues urgently needed, and titles especially useful in duplicate:

SPECIAL NUMBERS NEEDED

American Chemical Society. Journal. v. 68, no. 11, Nov. 1946
American Journal of the Medical Sciences. v. 213, no. 2, Aug. 1946
Anesthesiology. v. 4, 1943 (complete or any numbers)
v. 6, no. 4, July 1945
British Journal of Radiology. v. 16, 1943 (complete or any numbers)
Journal of Neuropathology. v. 6, no. 1, Jan. 1947
Journal of Neurosurgery. v. 3, no. 4, July 1946; and any numbers of 1946–1947
Medical Clinics of North America. v. 30, no. 5, 1946
Surgical Clinics of North America. v. 26, no. 4, 1946

DUPLICATES WELCOMED

American Journal of the Medical Sciences
American Medical Association. Journal

American Journal of Obstetrics and Gynecology
American Journal of Surgery
Annals of Internal Medicine
Annals of Surgery
Archives of Internal Medicine
Archives of Surgery
New England Journal of Medicine
Medical Clinics of North America
Southern Medical Journal
All state medical journals

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NOTICE OF REUNION AND ALUMNI DAY

Advance notice is given herewith of the program for the annual Medical Alumni Day.

This year the plans will include the presentation of the Alumni Honor Award for distinguished contribution to medical science by an outstanding alumnus of the Medical School There will be the usual Alumni reunions, a luncheon, the annual meeting of the Association, an interesting scientific program and the traditional Alumni banquet.

ALUMNI DAY WILL BE FRIDAY, JUNE 4, 1948

Class reunions this year will be held for the following classes: 1943; 1938; 1933; 1928; 1923; 1918; 1913; 1908; 1903; 1898. Representatives and class organizations will receive all possible aid in organizing class reunions by applying to the Alumni office.

Again, the Alumni office will secure hotel reservations for you in Baltimore upon proper application.

Further details of the program, reservation blanks for hotel accommodations and banquet tickets will be forwarded directly to you at a later date.

MEDICAL ALUMNI DAY—UNIVERSITY OF MARYLAND—JUNE 4, 1948

NEWS ITEMS

Dr. John C. Krantz, Jr., Professor of Pharmacology in the School of Medicine, delivered the annual B. A. Thomas Oration of the Philadelphia Urological Society on January 26, 1948. The subject of the address was "The Mechanism of the Action of the Anti-infective Drugs."

WILLIAM SISSON GARDNER, M.D.

William Sisson Gardner, Emeritus Professor of Gynecology, University of Maryland, passed away in the early afternoon of February 17, 1948. His passing in keeping with his life was quiet and unostentatious.

Born in Athens County, Ohio, September 23, 1861, the son of the Reverend Wilson and Emma Brown Gardner, he completed the primary grades of the public school, then graduated from Nelsonville, Ohio High School in 1878. Following this, he taught school in Ohio in order to raise funds for the medical education which he had long desired. He matriculated at the College of Physicians and Surgeons, Baltimore and graduated with distinction in 1885.

He then spent one year at the old Maternity Hospital on Lombard Street and the following year in company with Dr. Harry Friedenwald as resident at Mercy Hospital. During this period he was on the service of the late Dr. Thomas Opie. After completing his residency he entered practice, always devoting special attention to obstetrics and gynecology and gradually confining all his attention to gynecology.

In December 1897, he married Mary A. Maslin of Kent County, Maryland, who died in 1947, a few months before their fiftieth wedding anniversary.

Dr. Gardner's outstanding characteristics were his simplicity, directness, unswerving honesty and clear-sighted evaluation of facts—traits which inevitably led to minor conflicts with his associates but never detracted from their respect and admiration. It is worthy of comment that few, if any, who came under his guidance were ever other than devoted friends.

During his active years, Dr. Gardner contributed regularly and generously to contemporary medical literature, more than fifty-four monographs in all, the first in 1887, the last in 1938, any of which are well worth perusal by serious students.

In addition to this he published a textbook of gynecology in 1912.

He was among the earliest members of the American College of Surgeons. For a number of years he served as Treasurer of the Medical and Chirurgical Faculty and became President of that body in 1921.

Always consistent in his philosophy, Dr. Gardner was a life-long member of the Methodist Church and for many years served as a trustee of Mt. Vernon Church and also of the Asbury Foundation.

He is survived by two sons, William M. and W. Carville, one daughter, Mrs. C. Ridgely Howard and his devoted sister, Mrs. Blanche G. Powell.

Society in general and medicine in particular are the better for his consistent, simple, honest life.

Thomas K. Galvin, M.D.

DR. G. H. HOCKING

Dr. G. H. Hocking died on October 25, 1947, after having been bedridden for a year. He was 92 years old and was believed to be Maryland's oldest physician. He gave up his practice several years before his death, but continued to accept a few of his old patients.

Dr. Hocking was a graduate of the School of Medicine of the University of Maryland in 1879, and practiced medicine for a total of 67 years. He was a member of the American Medical Association and the Medical and Chirurgical Faculty of Maryland, and served twice as president of the Baltimore County Medical Society.

WILLIAM TARUN, M.D.

By the passing of William Tarun, Baltimore and the medical profession lost an excellent physician, a true friend and a genial companion. His memory will live in the hearts of his friends and patients for many years.

Dr. Tarun graduated from the School of Medicine of the University of Maryland in 1900. Shortly thereafter, he began work under Dr. J. Frank Crouch in the ear clinic at the Presbyterian Eye, Ear, Nose and Throat Hospital. Later he transferred to Dr. Hiram Woods' Eye Clinic, where he remained for a number of years. He was appointed chief of the Eye Clinic at the University of Maryland Hospital, and he held this post until Dr. Woods' retirement from the Department.

In his early professional career Dr. Tarun had a large practice in otology and mastoid surgery, but later he limited his work to ophthalmology.

With his keen mind and good training, he soon became one of the city's leading ophthalmologists. In 1917, he was elected a member of the American Ophthalmological Society, where he took a prominent part in the meetings, especially in the discussions. He contributed a few papers, but did not write extensively. Dr. Tarun was also a member of the American Academy of Ophthalmology and Oto-Laryngology, the Association for Research in Ophthalmology, the American College of Surgeons and the American Medical Association.

Although he retired from practice after the illness of Mrs. Tarun, he maintained an active interest in medicine. Outside his profession, his chief hobby was the raising of many kinds of flowers, which he cared for himself; in fact his fatal illness began while he was working in his garden.

OBITUARIES

Bacon, Walter Compton, Baltimore, Md.; class of 1911; aged 61; died, July, 1947.

Birely, Morris Allen, Thurmont, Md.; class of 1894; aged 75; died, October 5, 1947, of cerebral hemorrhage.

Duncan, Edward Montgomery, Baltimore, Md.; class of 1884; aged 86; died, September 29, 1947, of arteriosclerotic heart disease and bronchopneumonia.

Ellis, Alfred Lee, Baltimore, Md.; P & S, class of 1904; aged 78; died, September 11, 1947, of cerebral thrombosis and hypertension.

Fitzpatrick, Edward Francis, Newark, N. J.; B.M.C., class of 1903; aged 66; served during World War I; died, August 18, 1947, of heart disease.

Goodall, Edwin Baker, Boston, Mass.; class of 1909; died, August 6, 1947.

Holcomb, John Hampton, Hepburn, O.; P & S, class of 1892; aged 81; died, October 23, 1947, of coronary thrombosis.

Kennedy, Eugene Augustine, Woonsocket, R. I.; aged 75; died, August 17, 1947, of arteriosclerotic heart disease.

King, Oscar Wentworth, Coral Gables, Fla.; class of 1907; aged 63; served during World War I; died, October 16, 1947, of coronary artery disease.

Markens, Edward Wasgate, Newark, N. J.; B.M.C., class of 1905; aged 69; veteran of the Spanish-American War and World War I; died, August 4, 1947, of coronary thrombosis and hypertrophied prostate.

McClung, William Dennis, Huntington, W. Va.; class of 1907; aged 71; served in U. S. Public Health Service during World War I; died, November 2, 1947, of injuries received in a fall from a moving automobile at Dickson.

McDowell, Robert J., Auburn, N. Y.; P & S, class of 1911; aged 69; died, August 25, 1947, of multiple myeloma.

Medders, Charles Hamilton, Still Pond, Md.; class of 1894; aged 75; died, October 6, 1947, of carcinoma.

Nevin, Bruce Emerson, Morristown, N. J.; class of 1894; aged 79; died, August 14, 1947, of cerebral hemorrhage, hypertension and cirrhosis of the liver.

Nowell, Luther Alpheus, Colerain, N. C.; class of 1894; aged 78; died, August 11, 1947, of cerebral hemorrhage.

O'Neill, Joseph B., Pawtucket, R. I.; class of 1900; aged 75; died, November 1, 1947.

Orr, Hugh Montgomery, La Salle, Ill.; P & S, class of 1895; aged 80; died, November 16, 1947, of heart disease.

Paton, Thomas Lloyd, East Paterson, N. J.; P & S, class of 1887; aged 94; died, October 6, 1947, of hypostatic pneumonia.

Porter, Minor Gibson, Baltimore, Md.; class of 1886; aged 82; died recently of a fractured right hip as the result of a fall, bronchopneumonia and diabetes mellitus.

Routson, Thomas Clyde, Frederick, Md.; class of 1899; aged 74; died recently of pneumonia following an operation for carcinoma of the bowel.

Savin, Thomas Littleton, Little Rock, Ark.; class of 1897; aged 72; died, October 3, 1947, of lymphatic leukemia.

Shatswell, James Arthur, Beverly, Mass.; P & S, class of 1890; aged 83; died, August 26, 1947, of arteriosclerotic heart disease.

Solt, William J., San Pierre, Ind.; P & S, class of 1896; aged 77; died, August 15, 1947, of cachexia following a fractured hip received in a fall.

Whitaker, Joel, Indianapolis, Ind.; class of 1910; aged 70; died, October 13, 1947, of cardiac failure and pulmonary edema.

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